A COMPARATIVE EVALUATION OF RADIOLOGIC AND CLINICAL SCORING SYSTEMS IN EARLY PREDICTION OF SEVERITY IN ACUTE PANCREATITIS

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CERTIFICATE

This is to certify that the dissertation entitled ***"A COMPARATIVE EVALUATION OF RADIOLOGIC AND CLINICAL SCORING SYSTEMS IN EARLY PREDICTION OF SEVERITY IN ACUTE PANCREATITIS"*** is the bonafide work done by ***Dr. S. VIJAY RAJ,*** Post Graduate student (2011 – 2014) in the Department of General Surgery, Government Stanley Medical College and Hospital, Chennai under my direct guidance and supervision, in partial fulfillment of the regulations of The Tamil Nadu Dr. M.G.R Medical University, Chennai for the award of M.S., Degree (General Surgery) Branch - I, Examination to be held in April 2014.

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DECLARATION

I, **DR. S. VIJAY RAJ** solemnly declare that this dissertation titled "***A COMPARATIVE EVALUATION OF RADIOLOGIC AND CLINICAL SCORING SYSTEMS IN EARLY PREDICTION OF SEVERITY IN ACUTE PANCREATITIS***" is a bonafide work done by me in the Department of General Surgery, Government Stanley Medical College and Hospital, Chennai under the supervision of my unit chief **Prof. R.V SURESH, M.S.,** with the guidance of **PROF. P. DARWIN, M.S.,** and my Head of the Department **PROF. K. KAMARAJ, M.S.**

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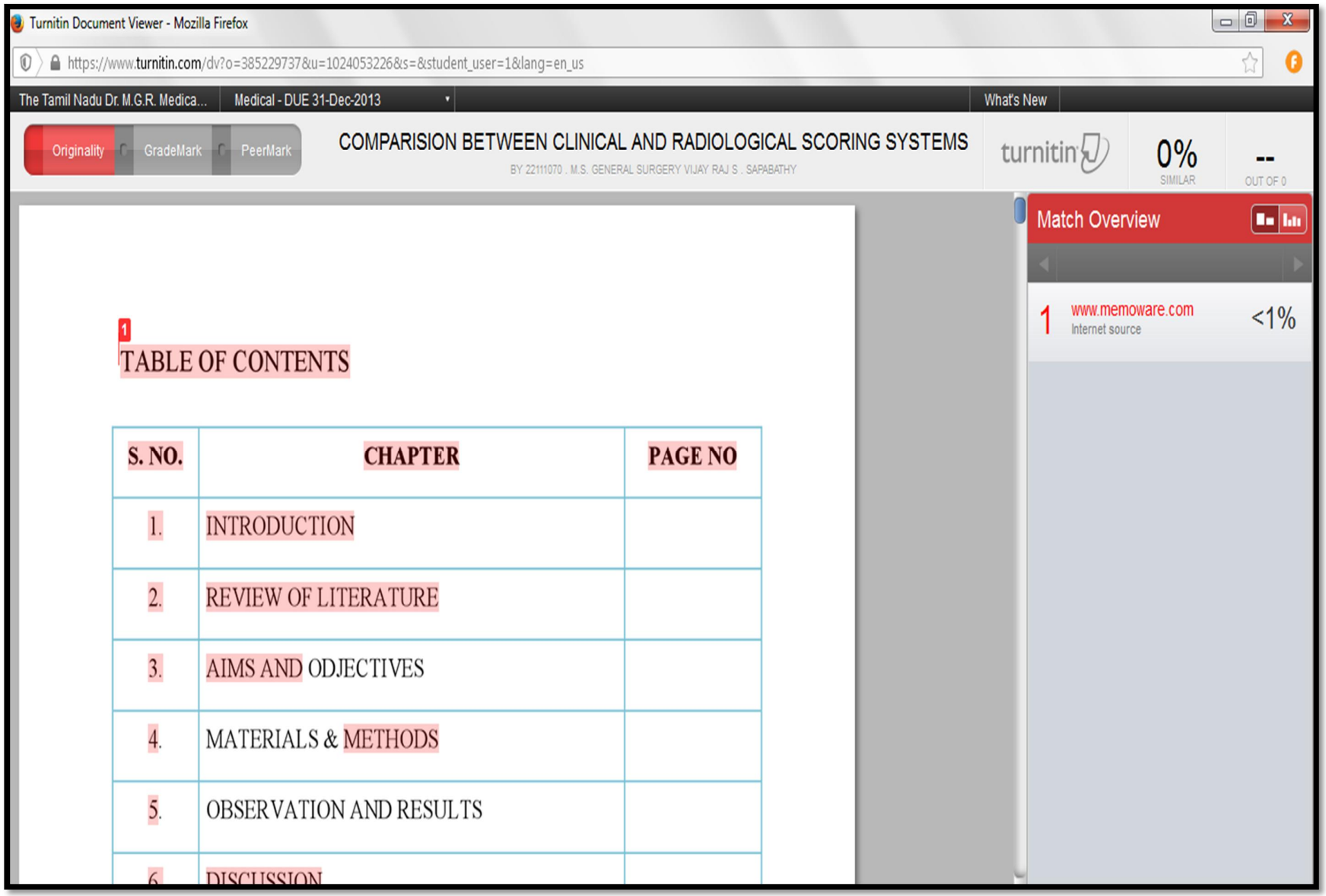


1

TABLE OF CONTENTS

TABLE OF CONTENTS

ABSTRACT

TITLE: ***A COMPARATIVE EVALUATION OF RADIOLOGIC AND CLINICAL SCORING SYSTEMS IN EARLY PREDICTION OF SEVERITY IN ACUTE PANCREATITIS***

AUTHOR: DR. S. VIJAY RAJ M.S POST GRADUATE

KEYWORDS: Pancreatitis, Bisap, Glasgow, Ct Severity Index, Organ Failure,

BACKGROUND: Demographic, clinical, and laboratory data of all consecutive patients with a primary diagnosis of ACUTE PANCREATITIS during a one year period is prospectively collected for this study. A retrospective analysis of the abdominal CT data is performed. CT severity index as well as two clinical scoring systems: Glasgow criteria / IMRIE'S prognostic criteria and Bedside Index for Severity in Acute pancreatitis (BISAP) were comparatively evaluated with regard to their ability to predict the severity of acute pancreatitis on admission (within 48 h of hospitalization).

First 50 patients attending the surgical emergency ward with clinical features of Acute Pancreatitis are evaluated clinically and subjected to laboratory and radiological investigations as per the designed

proforma. Data pertinent to the scoring systems will be recorded within 48hr of admission to the hospital. Once diagnosis is established the patient disease severity will be assessed by following the scoring systems CT SEVERITY INDEX, MODIFIED GLASGOW, BISAP. The accuracy of each imaging and clinical scoring system for predicting the severity of AP was assessed using appropriate statistical tools.

RESULTS: On keeping the cut of value for BISAP as 3, GLASGOW as 3 AND CTSI as 4 and analyzing using PEARSON CORRELATION it was found BISAP had 82.6% correlation compared to GLASGOW and CTSI which only had 51.4% correlation. If BISAP score predicts the disease to be severe then there is 82% positivity that the patient will have acute severe pancreatitis. In CRAMER V test the strength of association was found to be 0.826 for BISAP score which is very high for predicting complications. In other words only 23.6% of people with negative BISAP score will have complication. The strength of association for Glasgow and CTSI was 0.514 which is moderate association and there is 64.7% chance of false negativity with these scoring systems.

CONCLUSION: From this study, we conclude that the BISAP score could be a simple and better clinical scoring system for the evaluation of disease severity in acute pancreatitis than GLASGOW and CTSI.

INTRODUCTION

ACUTE PANCREATITIS is a reversible pancreatic parenchymal injury with inflammation which presents with varied clinical presentation, it mostly presents as mild self limiting disease but in about 10 – 20% of cases it presents with systemic complications which require intensive care unit treatment or surgical interventions and mortality in these cases can be as high as 30-40%. Severe acute pancreatitis is now found to be bi-phasic with systemic inflammatory response syndrome (SIRS) leading to Multi organ dysfunction syndrome (MODS) in the initial week which if resolutes by natural defences or treated by therapeutic intervention leads on to local pancreatic complications like pancreatic necrosis, sepsis and MODS in the ensuing weeks.

Practically we need to identify those patients who are more likely to develop complications of pancreatitis and this led to the development of scoring systems based on clinical and imaging criteria's. The rationale behind these scoring systems is to identify those high risk patients and manage them appropriately.

Scoring system in pancreas has been evolving ever since the development of Ranson's criteria in 1974. The other scoring system used are Modified Glasgow scoring system, Acute physiology and chronic health evaluation (APACHE I & II), Bedside index for severity of acute pancreatitis (BISAP) and Balthazar grading and CT severity index.

REVIEW OF LITERATURE

TABLE 1: LANDMARKS IN HISTORY OF PANCREAS

Herophilus	334B.C	First person to document the existence of pancreas
Rufus	100 A.D.	Considered pancreas as part of omentum and coined pancreas meaning all flesh
Galen		Described blood supply of pancreas
Massa	1536	Suggested pancreas as a cushion for stomach to rest on
Vesalius	1541	Proved that Galen description of pancreas was correct and showed pictorial illustrations of pancreas
Wirsung		Main pancreatic duct named after him
Vater	1720	Explained about the anatomy of duodenal papillae
Santorini	1724	Described accessory pancreatic duct along with other ducts
Morgagni	1769	Pancreatic malignancy – described adenocarcinoma
Soemmering	1791	Called it abdominal salivary gland
Treitz	1853	Identified trietz band and retropancreatic fascia
Langerhans	1869	Identified small round cells scattered throughout pancreas which was later named after him

MacBurney	1878	Removed calculi by doing duodenotomy
Von Winiwarter	1882	Pancreatic adenocarcinoma – first person to operate
Oddi	1887	Illustrated duodeno-panctreatic ampulla
Fitz	1889	Pathophysiology of pancreatitis
Bayliss and Starling	1902	Discovered secretin
Kocher	1903	Described surgical method of duodenal mobilization
Fabozzi	1903	Explained tumours of islet cells
Kausch	1909	Performed the first successful pancreatoduodenectomy
Banting and Best	1922	Isolated "insuline" from islet secretions of dog pancreas
Elman	1927	Invented the serum amylase test
Whipple	1930	Offered his triad in insulinoma: 1) symptoms of hypoglycemia during fasting; 2) serum glucose less than 50 mg/dL; 3) with administration of exogenous glucose the hypoglycemic symptoms disappear
Whipple	1940	Performed a one-stage excision of the entire head of the pancreas with total duodenectomy with 10-year survival
Rockey	1943	Performed the first total pancreatectomy
Doubilet and Mulholland	1965	Advocated sphincterotomy to treat acute pancreatitis

Kelly and Lillehei	1966	First clinical pancreas transplant
Fortner	1973	Described regional pancreatectomy
Traverso and Longmire	1978	Introduced the pylorus-preserving pancreatoduodenectomy
Beger et al.	1988	Described necrosectomy in management of necrotizing pancreatitis

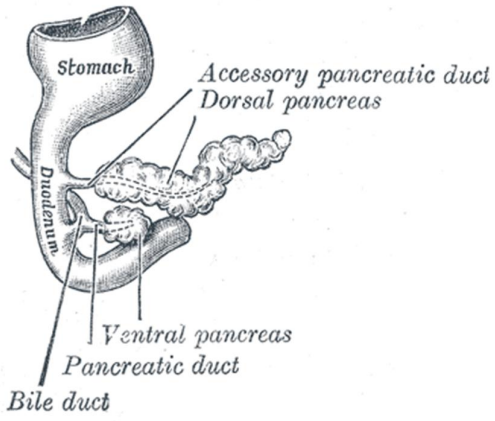
EMBRYOGENESIS OF PANCREAS

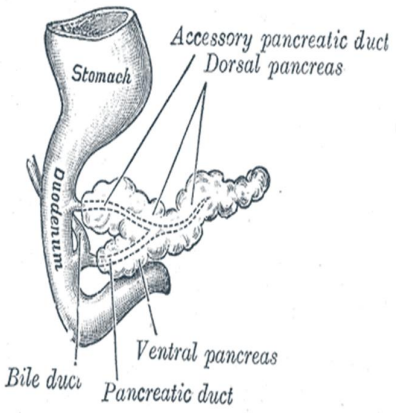
Genesis of pancreas begins at the end of fourth week of intrauterine gestation. It develops from the dorsal and the ventral primordium. The ventral primordium forms the duct of Wirsung, part of uncinate process and the head. The dorsal primordium forms duct of santorini, remainder of uncinate process and the body and tail. Both the dorsal and ventral primordium fuse at the sixth week. Secretion of insulin begins at fifth month.

Failure of fusion of dorsal and ventral pancreatic buds results in Pancreatic divisum. In this condition main and accessory pancreatic duct drain separately. Pancreatitis is a common complication of this anomaly.

Annular pancreas is another anomaly of pancreas where second part of duodenum is surrounded by a band of pancreatic tissue causing

stenosis at that level. Other congenital conditions include Heterotrophic pancreatic tissue, Accessory pancreas and pancreatic gall bladder.



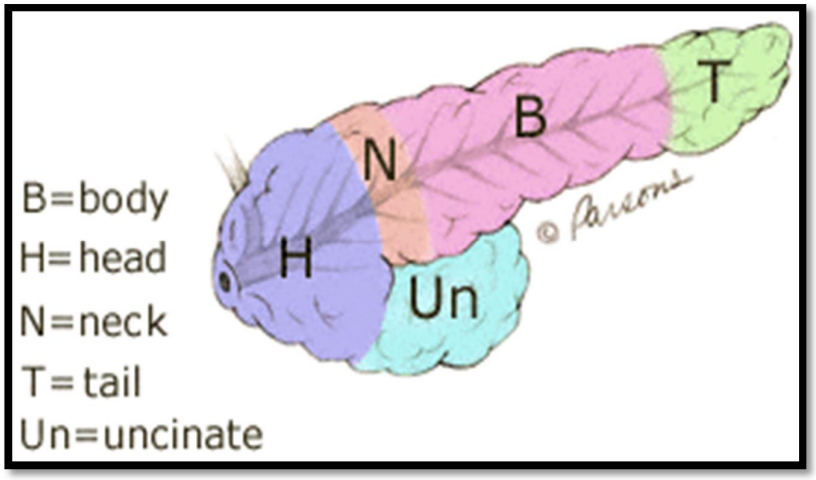


SURGICAL ANATOMY OF PANCREAS

Pancreas is one of the treacherous organs to operate on because often its location. It is a retroperitoneal organ which is closely related to the duodenum stomach, spleen, transverse mesocolon, great vessels and omental bursa.

Pancreas can be divided into four parts head, neck, body and tail. Head of pancreas is flattened antero-posteriorly with its anterior surface related to pylorus and transverse colon and the posterior surface related to right kidney, right crus of diaphragm, inferior vena cava, and right gonadal vessels. Head of pancreas is lies adherent to C loop of duodenum.

The word uncinate means hook like. It is a projection from the head of pancreas with highly variable anatomy. On cut section, it is located between aorta and superior mesenteric vessels with left renal vein above and third part of duodenum below.



The pancreatic tissue between the passage of superior mesenteric vessels and the beginning of portal vein dorsally is the neck of pancreas which is 1.5 to 2 cm long. It is anteriorly related to the gastroduodenal artery giving rise to superior pancreatico-duodenal artery and posteriorly related to portal vein formed by confluence of superior mesenteric and splenic vein.

Body of pancreas is related to the lesser omentum of stomach and transverse mesocolon anteriorly and posteriorly related to origin of superior mesenteric artery, left renal and suprarenal gland, and splenic vein.

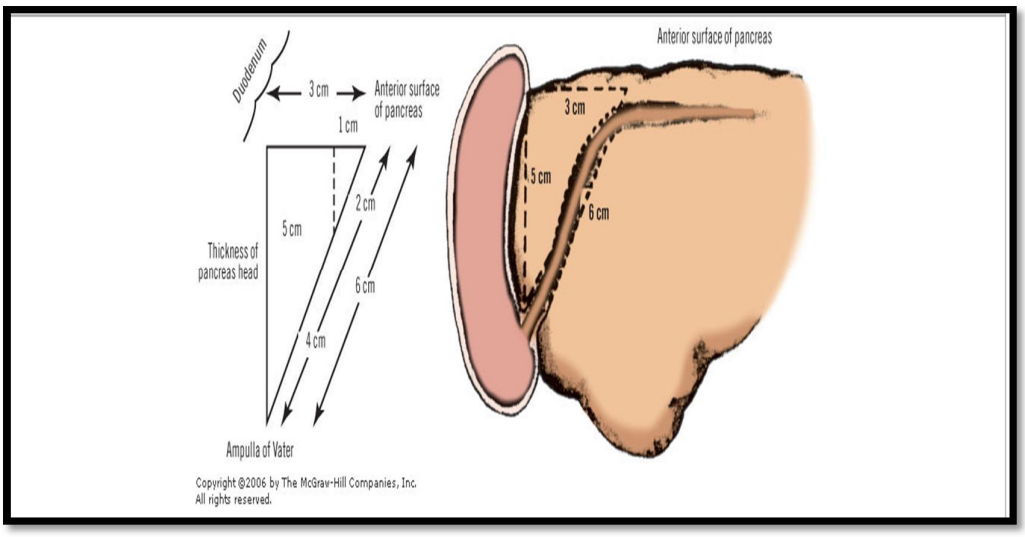
The tail is related to hilum of spleen and is enclosed along with splenic artery and splenic vein in splenorenal ligament.

PANCREATIC DUCTAL ANATOMY

The duct of wirsung starts from the tail and is situated midway between superior and inferior margins and more posterior than anterior. In about 50% of cases it crosses the first lumbar vertebrae. The tributaries enter at right angles to the main pancreatic duct in tail and body of pancreas and the superior and inferior pancreatic tributaries alternate with each other. The approximate number of tributaries is 15-20.

The main duct turns caudal and posterior at the level of head of pancreas and on reaching major papillae it becomes horizontal and joins posterior surface of CBD and enters wall of duodenum at the level of second lumbar vertebra. The diameter of the duct varies from head to tail. Usually it is largest at the head and narrowest at the tail. It is approximately 3mm at the head, 2mm in body and 1mm in tail of pancreas.

The accessory duct of santorini drains the superior portion of head and is smaller in size than the main duct. It usually drains into minor duodenal papillae but can sometimes join the main duct.



PAPILLA OF VATER AND AMPULLA OF VATER

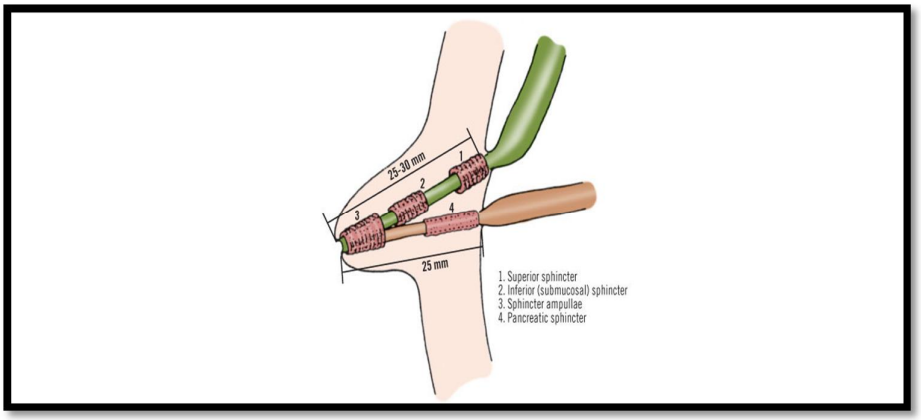
The major duodenal papilla otherwise known as papilla of vater is a mound like projection in the second part of duodenum through which the distal portion of ampulla passes into duodenum. Bidloo in 1685 first illustrated papilla but the structure bears the name of Abraham Vater.

The papilla of vater is located posteromedially approximately at the level of second lumbar vertebrae in the second portion of duodenum. Endoscopically it can be identified where longitudinal and transverse folds meet forming a T-shaped mucosal fold.

Ampulla is the name given to the dilatation of the common pancreaticobiliary channel. There can be three variations in ampulla anatomy, they are

1. Pancreatic duct opens into CBD at a variable distance from papilla with or without dilatation
2. Pancreatic and bile duct open near one another into major duodenal papillae
3. Pancreatic and duodenal papillae open separately into duodenum

The minor papilla is 2cm superior to the major papilla and the landmark for identification is gastroduodenal artery. The blood supply of ampulla of vater is from the posterior superior pancreaticoduodenal arteries (PSPD) and from arcade arising from postero superior and posteroinferior pancreatic arteries.

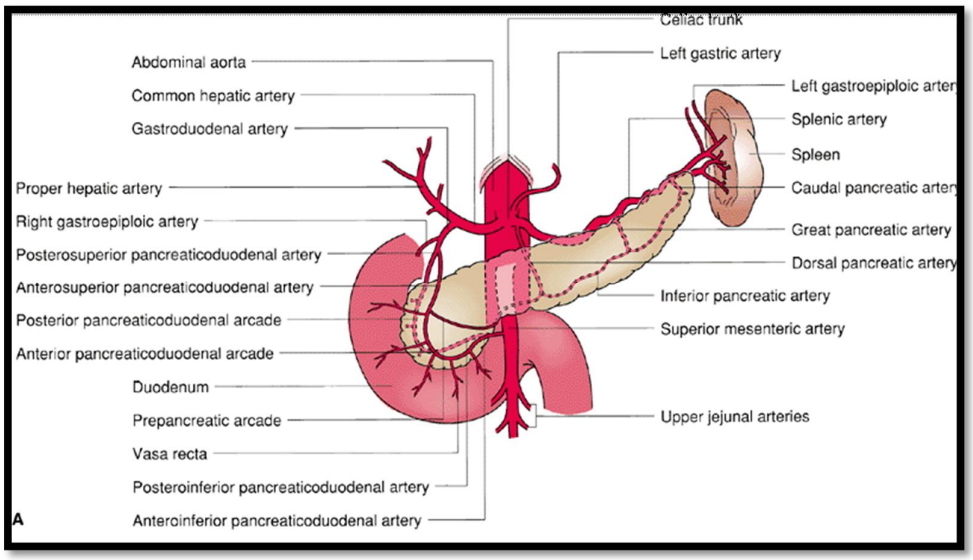


The sphincter of boyden includes smooth muscle fibers from intramural part of bile duct, pancreatic duct, ampulla and from duodenal musculature. The sphincter guarding the ampulla is cut during

sphincterotomy at 11’0 clock position to avoid damage to vessels. The vessels that can get commonly damaged are retroduodenal and anamolous right hepatic artery. To avoid injuring it is better to palpate posteriorly and look for any pulsation, which if present will indicate anomalous artery.

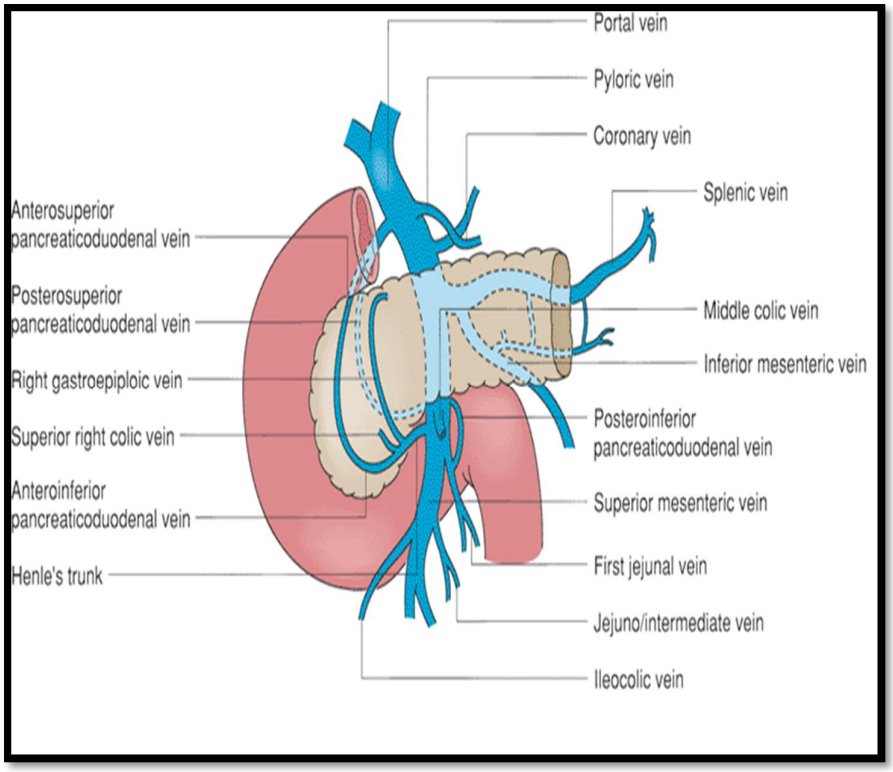
BLOOD SUPPLY OF PANCREAS

The blood supply of pancreas is complex, variable and atypical. The arterial supply of pancreas comes from both celiac plexus and splenic artery of which the splenic artery forms the major blood supply. Anterior and posterior superior pancreatico duodenal artery arising from gastroduodenal artery anastamose with anterior and posterior inferior pancreatico duodenal artery arising from superior mesenteric artery. This anastamotic arcade supplies the head of pancreas and duodenal wall.



The gastroduodenal artery which is a branch of common hepatic artery gives rise to supraduodenal , gastroduodenal and superior pancreatico duodenal arteries. The anterior superior pancreatico duodenal (ASPD) artery gives eight to ten branches to anterior surface of pancreas and anastamose with anterior inferior pancreatico duodenal artery. Supraduodenal and retroduodenal arteries supply the first part of duodenum. The posterior arcade lies away from duodenum posterior to lower end of Common bile duct. Injury to the ASPD can occur during puestow procedure.

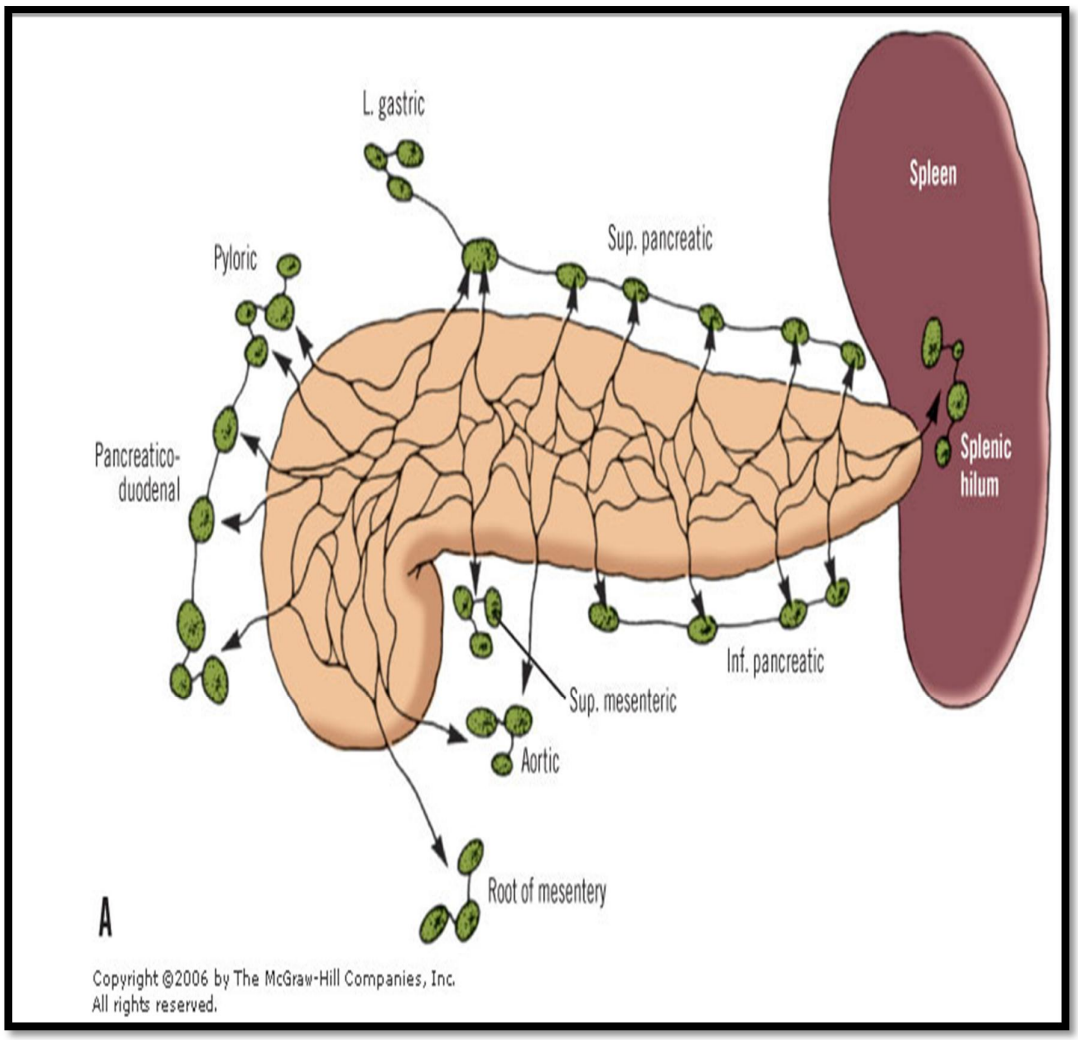
The splenic artery gives rise to dorsal pancreatic artery which in turn gives origin to transverse or inferior pancreatic artery, the great pancrteatic artery or pancreatic magna and the caudal pancreatic artery sometimes. The course of splenic artery above the body and tail pancreas is tortuous. Failure of anastamosis of inferior pancreatic artery with gastroduodenal artery can cause necrosis of tail if blocked by emboli. Pancreatic surgeon should be aware of anomalous hepatic arteries and anomalous middle colic arteries during surgery.



The venous drainage of pancreas follows the arterial supply and they lie superficial to the artery. The drainage is to the portal vein, splenic vein, superior and inferior mesenteric veins. Both artery and vein lie posterior to the ducts. There are usually no branches on the anterior surface of portal vein.

LYMPHATIC DRAIANGE OF PANCREAS

The lymphatics from pancreas drain into five main groups which are anterior, posterior, superior, inferior and splenic nodes. The superior group drains from upper half of head and body of pancreas. The inferior group drains the lower half of head and body of pancreas. The splenic group predominantly drains tail of pancreas.



NERVE SUPPLY

Pancreas gets both sympathetic and parasympathetic innervations. The sympathetic innervations are from preganglionic greater and lesser thoracic splanchnic nerves which relay into celiac and superior mesenteric ganglion. Postganglionic branches from this ganglia supply pancreas by accompanying major blood vessels. The parasympathetic innervations is via the vagus.

HISTOLOGY AND PHYSIOLOGY OF PANCREAS

Pancreas is a mixed exocrine and endocrine gland which does not have a definitive capsule but surrounded by fine connective tissue. The islets of langerhans are scattered throughout pancreas (constitute 2% of gland tissue). The exocrine portion of pancreas is composed of dark staining acini which are in arranged as tubular and spherical masses forming subunits of lobule.

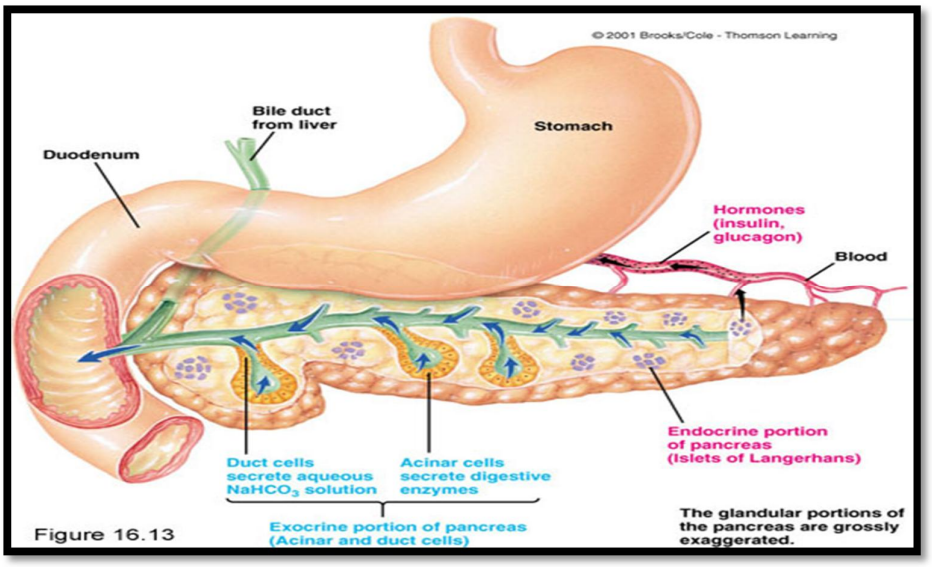
In response to a meal the acinar cells undergo cyclical changes in morphology. With the help of electron microscopy subcellular structures in acinar cell can be studied. The acinar cells has short slender microvilli extending into lumen of acinus. The golgi complex which plays an important role in the formation of zymogen granules and the transport of secreting proteins is situated between nucleus and the mass of zymogen granules in resting gland. There are two types of secretory granules in pancreas, electron dense zymogen granules and electron lucent condensing vacuoles. Zymogen granules are membrane bound spherical vesicles containing the digestive enzymes. The functional unit of exocrine pancreas is the acinus and its draining ductile which in turn joins the intercalated ducts which finally drains into main pancreatic duct.

Exocrine pancreatic secretions include both inorganic and organic secretions. The inorganic secretion constitutes water, sodium, potassium, bicarbonate and chloride. The average secretion is around 800 to 1000ml per day. The flow rate increases from 0.3ml/minute to 4.0 ml/minute during meal. Secretin stimulation causes increased volume of pancreatic fluid. Pancreatic enzymes originate in the acinar cells. Secretion of water and electrolytes originates in the centroacinar and intercalated duct cells.

Centroacinar cells and ductular epithelium secrete 20 mmol of bicarbonate per liter in the basal state. Major stimulants are Secretin, Cholecystokinin, Gastrin and Acetylcholine. Major inhibitors are Atropine, Somatostatin, Pancreatic polypeptide and Glucagon. Secretin is released from the duodenal mucosa in response to a duodenal luminal pH < 3.

Amylase is the only digestive enzyme secreted by the pancreas in an active form. It functions optimally at a pH of 7 and hydrolyzes starch and glycogen to glucose, maltose, maltotriose, and dextrins. Lipase functions optimally at a pH of 7 to 9 emulsify and hydrolyze fat in the presence of bile salts. Proteases are essential for protein digestion which are secreted as proenzymes and require activation for proteolytic activity. The duodenal enzyme enterokinase converts trypsinogen to trypsin.

Trypsin in turn, activates chymotrypsin, elastase, carboxypeptidase, and phospholipase .



The following table shows the various digestive enzymes secreted by pancreas.

TABLE 2 – DIGESTIVE ENZYMES OF PANCREAS

Proenzymes*	Cationic trypsinogen
	Anionic trypsinogen
	Mesotrypsinogen
	Chymotrypsinogen (A, B)
	Kallireinogen
	Procarboxypeptidase A (1, 2)
	Procarboxypeptidase B (1, 2)
	Prophospholipase

	Proelastase
Enzymes	Amylase
	Carboxylesterase
	Sterol esterase
	Lipase
	DNase
	RNase

The regulation of pancreatic enzyme secretion is mediated via both humoral and neural pathways. In addition to the enzymes pancreatic trypsin inhibitor is produced which prevents auto catalytic digestion of pancreas and henceforth pancreatitis.

There are about one million islet cells in the pancreas, each measuring 0.2mm in diameter and surrounded by a rich network of capillaries with fenestrated endothelium. The acinar cells surrounding the islets are called peri islet acinar cells which are biochemically different from tele acinar cells (located away from islet cells). Insulin Synthesized in the Beta cells of the islets of Langerhans is the only hormone reducing the blood glucose level. Proinsulin is transported from the endoplasmic reticulum to the Golgi complex where it is packaged into granules and cleaved into insulin and a residual connecting peptide, C peptide. Major inhibitors are somatostatin, amylin, pancreastatin and α-sympathetic

fibers. Major stimulants are Glucose, amino acids, glucagon, GIP, CCK, sulfonylurea compounds, β-Sympathetic fibers. 80% of the islet cell mass must be surgically removed before diabetes becomes clinically apparent.

Glucagon is secreted by the A cells of the islet. Glucagon elevates blood glucose levels through the stimulation of glycogenolysis and gluconeogenesis Major stimulants are Aminoacids, Cholinergic fibers and β-Sympathetic fibers. Major inhibitors are Glucose, insulin, somatostatin and α-sympathetic fibers.

D cells of the islet secrete somatostatin which inhibits the release of growth hormone and release of almost all peptide hormones. It also inhibits gastric, pancreatic, and biliary secretion. It is used to treat both endocrine and exocrine disorders.

ACUTE PANCREATITIS

Terms and definitions:

When acute it is often a mortal ill. It strikes the patient suddenly and often, strikes to kill. These are the words of Zachary cope regarding pancreatitis.

It is an auto digestion of the pancreatic and peripancreatic tissues resulting in local and systemic manifestations, many of which are catastrophic and fatal.

Mild acute Pancreatitis: Minimal organ dysfunction responsive to fluid administration. *Severe acute pancreatitis*: One of the following: Local complications (Pancreatic necrosis, Pancreatic pseudocyst, Pancreatic abscess), Organ failure, Ranson criteria >3 or APACHE II> 8 points.

Acute fluid collections: Fluid collections in or near the pancreas which occurs early in the course charaterized by lack a defined wall. *Pancreatic necrosis*: Non viable pancreatic tissue diagnosed by IV CECT. *Acute pseudocyst*: Fluid collection containing pancreatic secretions with a defined wall. *Pancreatic abscess*: Collection of pus usually in or near pancreas.

In general gastroenterologist uses the Atlanta classification to grade the severity of pancreas.

Atlanta Criteria for Severe Acute Pancreatitis

Organ Failure
a. Shock: systolic blood pressure <90 mm Hg
b. Pulmonary insufficiency: $Pao_2 \leq 60$ mm Hg

c. Renal failure: serum creatinine >2 mg/Dl

d. Gastrointestinal bleeding: >500 mL/24 hr

Local Complications

a. Necrosis

b. Abscess

c. Pseudocyst

Unfavorable Early Prognostic Signs

a. Ranson's signs (see Table 58-2)

b. APACHE-II points

NATURAL HISTORY:

The disease process seems to involve two phases. The first phase is related to the inflammatory cascade that usually lasts for a week. During this phase extrapancreatic organ failure secondary to systemic inflammatory response is elicited by acinar cell injury. Infection is rare in this phase. Fever, tachyacardia, hypotension, tachypnoea, and leukocytosis are typically seen. This phase can resolute with some amount of pancreatic edema or progress to irreversible liquefactive necrosis or form fluid collections in and around pancreas. The severity of organ failure and the extent of pancreatic or peripancreatic involvement is directly propotional.

Nearly 25% of cases develop a more protracted disease course without undergoing resolution. The second phase of pancreatitis is the development of necrotizing pancreatitis. This phase is complicated by sepsis and multiorgan failure. The mortality in pancreatitis is common during first week and later during the third week due to infection in pancreatic necrosis. Mortality is higher in older, comorbid patients than younger population.

PATHOPHYSIOLOGY:

The pathophysology of pancreatitis on the basis of Autodigestion theory suggests that proteolytic enzymes are activated within the pancreas rather than in the intestinal lumen. The trypsin enzyme activated causes a cascade of other enzymes to be activated initiating pancreatitis. Normally there are certain intrapancreatic mechanisms causing inactivation of trypsin, they are the pancreatic secretory trypsin inhibitor now known as SPINK 1, mesotrypsin, peptide-y and trypsin itself. They are also certain non specific proteases like alpha-1-antitrypsin and alpha-2-macroglobulin. When the amount of activated trypsin overhelms the defence mechanism pancreatitis ensues. Trypsin activated peptide concentration in urine and ascitis fluid correlates with the severity of pancreatitis.

INITIAL PHASE is characterized by acinar cell injury due to intrapancreatic digestive enzyme activation. Zymogen activation mediated by lysosomal hydrolases e.g. cathepsin B

SECOND PHASE constitutes Intrapancreatic inflammation reaction due to activation, chemoattraction, and sequestration of neutrophils in the pancreas. This neutrophil sequestration can activate trypsinogen

In the THIRD PHASE activated proenzymes, (esp. Trypsin) digest pancreatic and peripancreatic tissues and activate other enzymes (i.e. elastase, phospholipase) due to effects of activated proteolytic enzymes and cytokines, released by inflamed pancreas, on distant organs, most notably the lungs which may result to SIRS and ARDS, and Multiorgan failure.

The second theory of co-localization of enzymes states that co localization of pancreatic enzymes in lysosome causes acinar cell injury which leads to pancreatitis. Cathepsin b is responsible for co-localization of enzymes and inhibitor of Cathepsin b may prevent trypsinogen activation and therby pancreatitis. In experimental models the disruption of paracellular barrier of acinar and duct cells cause extravasation of enzymes into interstitial spaces causing interstitial edema.

The activation of the enzymes leads to microcirculatory injury, leukocyte chemoattraction followed by release of cytokines, free radical

production, pancreatic fluid accumulation and bacterial transmigration to pancreas leading to systemic sepsis. Microcirculatory changes are vasoconstriction with stasis and hence decreased oxygen saturation, and progressive ischemia.

This microcirculatory failure leads to release of proinflammatory cytokines such as Tumour necrosis factor, interleukin -1, 6, 8 and platelet activating factor. The next step is the formation of reactive oxygen species which further aggravates microcirculation leading to increased vascular permeability and henceforth thrombosis and haemmorhage ending up in pancreatic necrosis.

Systemic complications in pancreatitis include fever, ARDS, metabolic complications, pleural effusion, renal failure, myocardial depression and shock. The pancreatic enzymes (phospholipase, elastase, trypsin) and cytokines (tumour necrosis factor, platelet activating factor) which get released into portal circulation cause systemic inflammatory response syndrome. The cytokines on reaching liver causes acute phase protein synthesis namely C-reactive protein and IL-6.

Acute respiratory distress syndrome is mainly due to phospholipase A which degrades the lung surfactant. Renal failure is a result of hypovolemia and hypotension. Metabolic complications are hyperlipidemia, hyperglycemia with or without ketosis, hypoglycemia

and hypocalcemia which in turn is mainly due to hypoalbuminemia , hypomagnesia and soap formation.

Infective necrosis and infective psuedocyst is mainly due translocation of bacteria from gut due breakdown of immunological barriers and ischemia of gut wall as a result of arteriovenous shunting of blood. Infection can also come via hematogenous route.

PREDISPOSING CONDITIONS

The number of conditions predisposing to pancreatitis is growing day by day

Metabolic:

- alcoholism
- hyperlipoprotienemia
- hypercalcemia
- drugs
- genetic

Mechanical:

- trauma
- iatrogenic injury
- endoscopic procedure & perioperative injury.

Vascular:

- shock

- athero embolism
- polyarthritis nodosa
- SLE
- HSP

Infectious:

- mumps
- coxsackie virus
- mycoplasma

Obstruction of biliary duct:

- periampullary tumour
- gall stone
- pancreatic divisum
- choledochocele
- ascaris lumbricoides
- clonorchis sinensis

Genetic:

- cationic trypsinogen
- trypsin inhibitor

Less common causes

- Pancreas divisum
- Chinese liver fluke

- Ischemia (bypass surgery)
- Cystic fibrosis

OBSTRUCTIVE CAUSES

- *GALLSTONES*

Along with alcohol it forms one of the most common causes of pancreatitis. Only 3-7% of patients with gallstones get pancreatitis. More common in stones which are less than 5mm as they can pass through cystic duct and cause ampullary obstruction. Recurrence of pancreatitis can be prevented by cholecystectomy and clearance of stones from common bile duct.

- *BILIARY SLUDGE AND MICROLITHIASIS*

Stones less than 3mm are known as microlithiasis, which usually hide in a viscous suspension of bile called biliary sludge. Biliary sludge composed of calcium monohydrate or calcium bilirubinate is usually asymotomatic but association with pancreatitis is proven in some studies. It appears as a low amplititude echo on ultrasound without characteristic acoustic shadow of gall stone. Biliary sludge can be due to prolonged fasting, ceftriaxone administration and total parenteral nutrition. At present no consensus are present on treatment protocol for biliary sludge.

- *TUMOURS*

Intraductal papillary mucinous neoplasm is the most common tumout causing pancreatitis, pancreatic adenocarcinoma can also cause pancreatitis rarely.

- *OTHER OBSTRUCTIVE CAUSES*

Annular pancreas, choledochocele, duodenal diverticula, and parasites like ascaris and clonorchis obstruct the pancreatico-biliary system .

ALCOHOL, TOXINS AND DRUGS

The effects of ethyl alcohol are modulation of pancreatic exocrine secretion in such a way that lithogenicity of pancreatic juice increases and causes stone formation, Contraction of sphinter of oddi, direct toxic effects on acinar cell, Directly activating trypsinogen,and Oxitative stress / free radicles formation. Usually alcohol causes chronic pancretitis but episodes of acute pancreatitis can be seen. The fatty acid ester of ethyl alcohol is the toxic metabolite. Another hypothesis is the de novo fibrosis of pancreas which states that cytokines stimulate stellate cells which in turn cause periductal fibrosis and thereby ductal obstruction and stone formation. In acute pancreatitis due to alcohol if inciting factors are removed then pancreatitis resolves spontaneously.

Table 3 -- Drugs Associated with Acute Pancreatitis

- Acetaminophen
- Alphamethyldopa
- 5-Aminosalicylic acid compounds
- Sulfasalazine
- Azodisalicylate
- Mesalamine
- Carbimazole
- Cimetidine
- Clozapine
- Dapsone
- Dexamethasone
- Enalapril
- Erythromycin
- Estrogen
- Furosemide
- Hydrochlorothiazide
- Hydrocortisone
- Isoniazid
- Lamivudine
- Losartan
- Metronidazole
- Nelfinavir
- Simvastatin
- Sulfamethazole
- Tetracycline
- Trimethoprim-sulfamethoxazole
- Valproic acid

Drugs can cause pancreatitis by three mechanisms hypersensitivity, Toxic metabolite and intrinsic toxicity.

METABOLIC DISORDERS

- *HYPERTRIGLYCERIDEMIA*

It is the third most common cause of acute pancreatitis after stone and alcohol. Serum triglyceride concentration greater than 1000mg/dl causes pancreatitis. It is Commonly seen in children with inherited hypertriglyceridemia and lipoprotein metabolism. Type I, II and V hypertryglyceridimia patients are more prone to attacks of pancreatitis and to prevent this lipoprotein should be less than 200mg/dl. Some of these patients may not manifest until an acquired trigger in the form of diabetes, alcohol or drugs is present.

- *HYPERCALCEMIA*

Increase in serum calcium can cause activation of trypsinogen within pancreatic duct and thereby pancreatitis. But usually hypercalcemia is rarely associated with pancreatitis and sometimes can occur in hypercalcemia due to hyperparathyroidism, metastatic bone disease, vitamin D toxicity and sarcoidosis.

- *INFECTIONS*

Radiological or tissue evidence of inflammation in pancreas is known as definite pancreatitis. Biochemical elevation of serum lipase or amylase

with symptoms is called probable pancreatitis and asymptomatic patients with only biochemical evidence are known as possible pancreatitis. Organisms associated with definite pancreatitis are Viruses (mumps, coxsackievirus, hepatitis B, , hepatitis A, hepatitis C, cytomegalovirus, varicella-zoster, herpes simplex and Epstein-Barr); bacteria (*Mycoplasma*, *Salmonella*, tuberculosis, *Legionella*, *Leptospira*, ,and brucellosis); fungi (*Aspergillus* and *Candida albicans*); and parasites (*Toxoplasma, Cryptosporidium, Ascaris, Clonorchis sinensis.* A Defienite criterion to define an organism as a cause of pancretitis is to do a culture or stain of organism in pancreas or duct.

- *VASCULAR DISEASE*

Ischemia of pancreas can occur due to vasculitis, emboli from atheromatous plaques, hemorrhagic shock, or after cardiopulmonary bypass. The end result of ischemia is mild pancreatitis or fatal necrotizing panctreatitis.

- *TRAUMA*

Pancreatitis can occur both due penetrating trauma and blunt trauma and the injury can range from contusion to transection of gland. In blunt injury the transaction commonly occurs at the place where the duct crosses the spine. The management depends on the extent of adjacent organ injury and ductal involvement or not.

Table 4 -- Factors That Increase the Risk of Post-ERCP Pancreatitis

Patient Related
Young age, female gender, suspected sphincter of Oddi dysfunction, recurrent pancreatitis, history of post-ERCP pancreatitis, normal serum bilirubin
Procedure Related
Pancreatic duct injection, difficult cannulation, pancreatic sphincterotomy, precut access, balloon dilation
Operator or Technical Related
Trainee (fellow) participation, nonuse of a guidewire for cannulation, nonuse of a pancreatic duct stent in high-risk procedures

Early recognition of post-ERCP pancreatitis can be done by serum amylase or lipase measurement. Lot of studies on drugs to cause relaxation of sphincter of oddi have not proven beneficial. Proposed methods to decrease the risk of pancreatitis are pancreatic stent placement, usage of guidewire for cannulation and avoiding precut sphinctrotomies.

- *POST-ERCP*

Acute pancreatitis is one of the feared complications of ERCP. The pathophysiology of post-ERCP acute pancreatitis is multifactorial and depends on factors like chemical, thermal, mechanical, enzymatic and hydrostatic.

- *CONGENITAL CAUSES*

Hereditary pancreatitis is a genetic disease that causes childhood pancreatitis and increased risk for pancreatic malignancy. Other controversial causes are pancreatic divisum and sphincter of oddi dysfunction.

CLINICAL FEATURES

Abdominal pain is the major symptom in pancreatitis and it varies from mild to severe constant pain which is steady and boring in character located in epigastrium and periumbilical radiating to the back, chest, flank and lower abdomen. Pain more intense on supine, relieved by sitting.Pain usually occurs after intake of alcohol. Other features are vomiting and fever (inflammatory mediators), Mild jaundice (cholangitis), Oliguria, hypoxia, acidosis, shock and dehydration. Erythematous skin nodules and in about 10-20% of patients- basilar rales, atelectasis and pleural effusion are also present.

Clinical signs include tachycardia, tachypnoea, tenderness, gaurding, rigidityand abdominal distension due to ileus or ascites. Grey turner's sign occurs due to enzymes seepage across retroperitoneum causing haemorrhagic spots and ecchymosis in the flanks. Cullen' sign is Ecchymosis and discolouration around umblicus (umblical black eye) and

fox sign is Ecchymosis and discolouration below the below inguinal ligament.

Some physical findings suggest a specific cause of acute pancreatitis. Hepatomegaly, spider angiomas, and palmar thickening favor alcoholic pancreatitis. Eruptive xanthomas and lipemia retinalis suggest hyperlipidemic pancreatitis. Parotid pain and swelling are features of mumps. Band keratopathy (an infiltration on the lateral margin of the cornea) occurs with hypercalcemia.

LAB INVESTIGATIONS

Serum amylase, more specifically isoenzyme- P is increased more than two to three fold in pancreatitis. Does not parallel the severity of attack, it rises in 2 to 6hrs, and declines after 3-6 days. Rising titre>1000 somogyi unit is significant. Limitation of serum amylase is that it is not 100% sensitive or specific. Urinary amylase remains elevated for longer period. Serum amylase can be falsely normal in hypertriglyceridemia-associated pancreatitis because of an amylase inhibitor.

Hyperamylasemia is not specific for pancreatitis and hence it is only a supportive tool in diagnosis. It is also elevated in salivary gland diseases, fallopian tube diseases, ovarian tumours, hollow viscus perforation and many more. Macroamylaemia is a condition where serum amylase is elevated but urinary amylase is normal. It is due to large amylase

molecules bound to immunoglobulins seen in circulation and not filtered through kidney. To rule out this urinary amylase to creatinine clearance ration is seen. Urine amylase/serum amylase x serum creatinine/urinary creatinine x 100. Normal value is 1-4%, >6% indicates acute pancreatitis. It is also helpful to differentiate munchausen syndrome.

Serum Lipase is found predominantly in pancreas but also in gastric, intestinal mucosa and liver. It is cleared by the kidney and hence renal failure will lead to elevated levels. Most appropriate cut-off is 2-3 x normal level. More accurate test than amylase, better specificity (90% vs. 75%)

Other parameters include Serum lactescence which is related to triglyceride metabolism and most specific in hypertryglycedimia. Serum trypsin is a more accurate indicator. Serum calcium is measured to detect hypocalcemia due to saponification. Trypsinogen activation polypeptide - serum &urine assay, phospholipaseA2, LDH, CRP>150 mg/l are all markers elevated in pancreatitis.

Routine investigations like Liver function tests, Renal function test, Blood sugar (Hyperglycemia), Total count, Haematocrit, Platelet count, Coagulation profile, Arterial PO2and PCO2- to assess pulmonary insufficiency (ARDS) should all be done. Peritoneal tap - high amylase, protein level and lipase level indicate pancreatic ascites.

Methemalbuminemia -indicate poor prognosis. Inflammatory mediators & acute phase reactants (IL1, IL6, TNF, CRP) predicts the severity of disease.

Apart from the battery of investigations mentioned above certain standard investigations should also be done. These include white blood cell count which is often markedly elevated in severe pancreatitis, blood glucose,Serum aspartate transaminase (AST), alanine aminotransferase (ALT), alkaline phosphatase, and bilirubin also may increase, particularly in gallstone pancreatitis. The erythrocyte mean corpuscular volume (MCV) tends to be higher in alcoholic patients and hence shown to help differentiate alcoholic from nonalcoholic acute pancreatitis. Serum triglyceride levels increase in acute pancreatitis, but also with alcohol use, uncontrolled diabetes mellitus, or defective triglyceride metabolism.

DIAGNOSTIC IMAGING

ABDOMINAL X- RAY

The findings on a plain radiograph vary from no abnormalities in mild disease to localized ileus of a segment of small bowel ("the sentinel loop") or the *colon cut-off sign* in more severe disease. Added advantage is an abdominal plain film helps exclude other causes of abdominal pain, such as obstruction and perforation.. Gastric abnormalities are caused by exudate in the lesser sac producing anterior displacement of the stomach,

thereby separation of the contour of the stomach from the transverse colon. Small intestinal abnormalities are due to exudate in proximity to small bowel mesentery and include ileus of one or more loops of jejunum (the sentinel loop), of the distal ileum or cecum, or of the duodenum. Generalized ileus may occur in severe disease.

Besides ileus, other abnormalities of the hollow GI tract may be present. The descending duodenum may be displaced and stretched by an enlarged head of the pancreas. In addition, spread of exudate to specific areas of the colon may produce spasm of that part of the colon and either no air distal to the spasm (the colon cut-off sign) or dilated colon proximal to the spasm. Head-predominant pancreatitis predisposes to spread of exudate to the proximal transverse colon, producing colonic spasm and a dilated ascending colon. Uniform pancreatic inflammation predisposes spread of exudate to the inferior border of the transverse colon and an irregular haustral pattern. Exudate from the pancreatic tail to the phrenicocolic ligament adjacent to the descending colon may cause spasm of the descending colon and a dilated transverse colon.

Other findings on plain radiography of the abdomen may give clues to etiology or severity, including calcified gallstones (gallstone pancreatitis), pancreatic stones or calcification (chronic pancreatitis with

a bout of acute inflammation), and ascites (severe pancreatitis). Gas in the retroperitoneum may suggest a pancreatic abscess.

CHEST RADIOGRAPHY

The findings on the chest roentgenogram occur in 30% of patients with acute pancreatitis, including elevation of a hemidiaphragm, pleural effusion(s), basal or plate-like atelectasis secondary to limited respiratory excursion, and pulmonary infiltrates. Pleural effusions may be bilateral or confined to the left side; rarely they are only on the right side. Patients with acute pancreatitis found to have a pleural effusion and/or infiltrate on admission are more likely to have severe disease. During the first 7 to 10 days, there also may be signs of congestive heart failure or acute respiratory distress syndrome. Pericardial effusion is rare.

ABDOMINAL ULTRASOUND

Abdominal ultrasonography is used during the first 24 hours of hospitalization to search for gallstones, dilation of the bile duct due to choledocholithiasis, and ascites. If the pancreas is identified (bowel gas obscures the pancreas 25% to 35% of the time), it is usually diffusely enlarged and hypoechoic. Less frequently there are focal hypoechoic areas. There also may be ultrasonographic evidence of chronic pancreatitis, such as intraductal or parenchymal calcification and dilation

of the pancreatic duct. Ultrasound is not a good imaging test to evaluate extrapancreatic spread of pancreatic inflammation or necrosis within the pancreas and consequently is not useful to ascertain severity of pancreatitis. During the course of acute pancreatitis, ultrasound can be used to evaluate progression of a pseudocyst (discussed later). Due to overlying gas, the diagnosis of cholelithiasis may be obscured during the acute attack but may be found after bowel gas has receded.

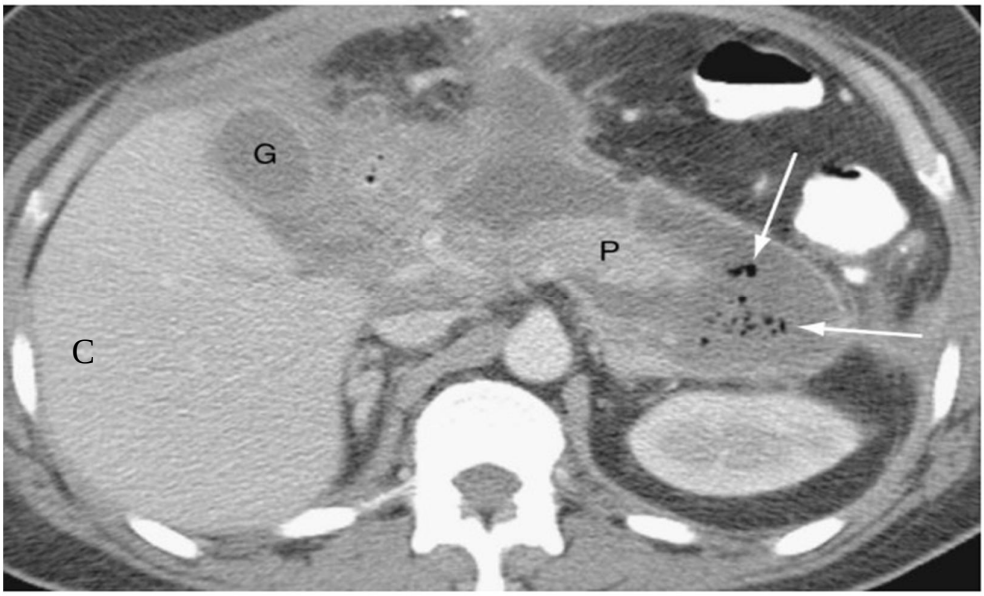
COMPUTED TOMOGRAPHY

CT is the most important imaging test for the diagnosis of acute pancreatitis and its intra-abdominal complications. The three main indications for a CT in acute pancreatitis are to exclude other serious intra-abdominal conditions, such as mesenteric infarction or a perforated ulcer; to stage the severity of acute pancreatitis; and to determine whether complications of pancreatitis are present, such as involvement of the GI tract or nearby blood vessels and organs, including liver, spleen, and kidney. Helical CT is the most common technique. If possible, scanning should occur after the patient receives oral contrast, followed by intravenous contrast to identify any areas of pancreatic necrosis. If there is normal perfusion of the pancreas, interstitial pancreatitis is said to be present. Pancreatic necrosis manifested as perfusion defects after intravenous contrast may not appear until 48 to 72 hours after onset of

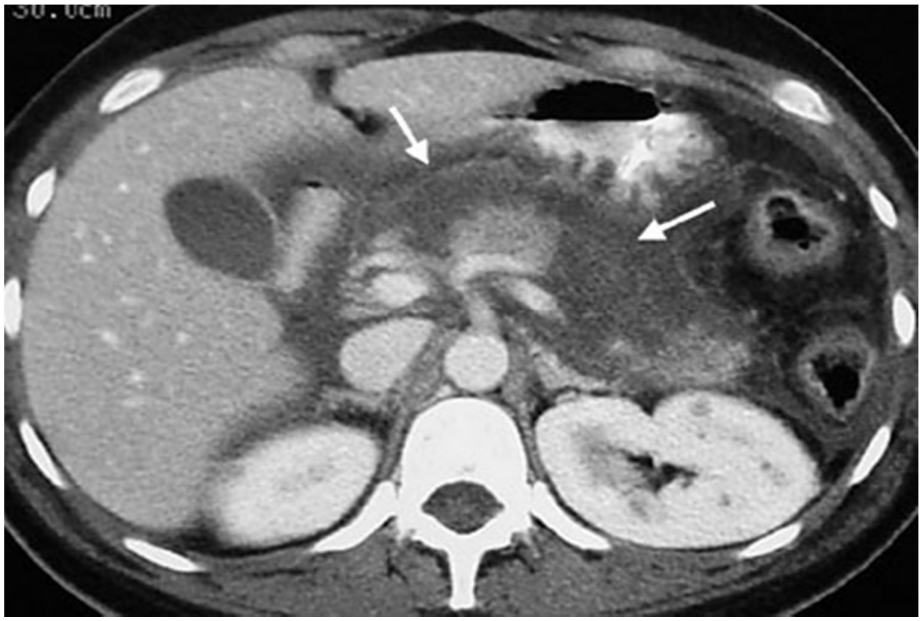
acute pancreatitis. Contraindications to using intravenous contrast are a patient's history of severe allergy (respiratory distress or anaphylaxis) or significant renal impairment (serum creatinine greater than 2 mg/dL). If severe renal impairment requires dialysis, intravenous contrast medium may be used. Hives or less severe allergic reactions with previous administration of iodinated contrast material are not absolute contraindications, but a nonionic contrast agent should be used, and 200 mg of hydrocortisone should be administered intravenously every six hours for four doses starting before the scan and 50 mg of diphenhydramine (Benadryl) should be given intramuscularly 30 minutes before the scan.

CT SHOWING EDEMATOUS PANCREAS WITH FLUID COLLECTION

CT SHOWING GAS POCKETS DUE TO STERILE NECROSIS



CT SCAN SHOWING NON- ENHANCING PANCREAS SUGGESTIVE OF NECROSIS



The severity of acute pancreatitis has been classified into five grades (A to E) based on findings on unenhanced CT. Although the presence of gas in the pancreas suggests pancreatic infection with a gas-forming organism, this finding can also accompany sterile necrosis with micro perforation of the gut or adjacent pseudocyst into the pancreas. Moreover, the great majority of pancreatic infections occur in the absence of gas on CT scan.

BALTHAZAR CT GRADING OF ACUTE PANCREATITIS

Prognostic Indicator	Points	Grade
Pancreatic inflammation		
Normal pancreas	0	A
Focal or diffuse enlargement of the pancreas	1	B
Intrinsic pancreatic abnormalities with inflammatory changes in peripancreatic fat	2	C
Single, ill-defined fluid collection or phlegmon	3	D
Two or more poorly defined collections or presence of gas in or adjacent to the pancreas	4	E
Pancreatic necrosis		
None	0	
≤ 30%	2	
> 30–50%	4	
> 50%	6	

MODIFIED CT SEVERITY INDEX

	Points
Pancreatic inflammation	
Normal pancreas	0
Intrinsic pancreatic abnormalities with or without inflammatory changes in peripancreatic fat	2
Pancreatic or peripancreatic fluid collection or peripancreatic fat necrosis	4
Pancreatic necrosis	
None	0
≤ 30%	2
> 30%	4
Extrapancreatic complications (one or more of pleural effusion, ascites, vascular complications, parenchymal complications, or gastrointestinal tract involvement)	2

ENDOSCOPIC ULTRASOUND

Usually endoscopic ultrasonography (EUS) is not helpful early in acute pancreatitis. Imaging of the pancreas during an attack of acute pancreatitis and weeks following an episode reveal signals that are not normal (typically hypoechoic) and indistinguishable from chronic pancreatitis and malignancy. However, after a month, especially in patients with idiopathic interstitial pancreatitis, EUS may help determine the presence of small tumors, pancreas divisum, and bile duct stones. EUS is equal to MRCP and ERCP but far more sensitive than either

abdominal ultrasonography or CT in detecting common duct stones. In a patient with biliary pancreatitis, whose serum bilirubin is rising in the setting of biliary sepsis, ERCP should not be delayed by first performing EUS. Although there has been some concern that ERCP can worsen pancreatitis in such settings, ERCP appears to be safe in acute pancreatitis if needed. One caveat is that the contrast instillation into the pancreatic duct could introduce infection into necrotic areas of the pancreas. For this reason, EUS might be the best method of evaluating the bile duct in a patient with necrotizing pancreatitis.

MAGNETIC RESONANCE IMAGING

MRI provides similar information regarding the severity of pancreatitis as does CT. MRI is as good as CT in detecting necrosis and fluid collections. MRI is better than CT, but equal to EUS and ERCP in detecting choledocholithiasis. The MRCP contrast agent gadolinium, previously thought to be safe in patients with renal failure, can cause nephrogenic systemic fibrosis (NSF), which has raised concern. MRI is less accessible and more expensive than CT. MRI also requires the patient to remain still during capture of images, which typically is longer than with spiral CT. The use of intravenous secretin prior to MRCP allows a better visualization of the pancreatic ducts. This has been shown

to be particularly useful in the evaluation of patients with idiopathic pancreatitis and recurrent pancreatitis.

PREDICTORS OF SEVERITY

Predicting severity of pancreatitis early in the course of disease is critical to maximize therapy and to prevent and minimize organ dysfunction and complications. Unfortunately the management of patients with acute pancreatitis is complicated by the inability to distinguish mild from severe disease during the early stages. The definition of the severity of acute pancreatitis early in the course of disease (during the first week) is typically based on clinical rather than anatomic parameters. At admission, several potential risk factors of severity and measurements that may reflect severity should be documented including age, body mass index, elevated hematocrit, elevated blood urea nitrogen (BUN), and pleural effusions or infiltrates on admission chest radiograph. The height of elevation of the serum amylase and lipase does not correlate with severity. Obese patients with pancreatitis have a higher incidence of local complications, respiratory failure, severe acute pancreatitis, and death from sterile necrosis than do nonobese patients.

Initially at presentation and over the first 48 hours, patients should be classified temporarily as having severe acute pancreatitis (and managed

as such initially) based on the presence of SIRS or organ failure. SIRS is defined by two or more of the following four criteria: pulse greater than 90 beats/minute; rectal temperature less than 36C or more than 38C; white blood count less than 4000 or more than 12,000/mm^3; and respirations greater than 20/minute or Pco_2 less than 32 mm Hg. The presence of SIRS at admission and persistence of SIRS to 48 hours increases the morbidity and mortality rate. In one study, 25% of patients with persistent SIRS died from acute pancreatitis, 8% with transient SIRS, and less than 1% without SIRS.

Although severity is now defined by the presence of organ failure or anatomic complications of acute pancreatitis, such as pancreatic necrosis, prospective systems using clinical criteria have been developed to determine severity in patients with acute pancreatitis. These systems include Ranson criteria and APACHE score. Unfortunately these scoring systems (discussed following) are cumbersome, requiring multiple measurements. Additionally, the systems are not accurate until 48 hours after presentation.

SCORING SYSTEMS

Ranson's Score

Ranson and colleagues identified 11 signs that had prognostic significance during the first 48 hours. The original list was analyzed in patients who primarily suffered from alcoholic pancreatitis and was then modified 8 years later for those with gallstone pancreatitis. Higher Ranson's scores predict more severe disease. In mild pancreatitis (scores < 2), the mortality is 2.5% and in severe pancreatitis (scores > 3) the mortality is 62%. Also, the higher the Ranson's score the higher the incidence of systemic complications, necrosis, and infected necrosis. These lists continue to remain in wide use in both the United States and Europe.

The Ranson criteria have several drawbacks. First, the list is cumbersome and there are two lists to follow depending on suspected etiology. Second, an accurate Ranson's score takes 48 hours to compute and the criteria have not been validated beyond the 48-hour time limit. Third, not all laboratories measure all the parameters in routine blood tests (e.g., serum lactate dehydrogenase [LDH]). Fourth, the overall sensitivity of the Ranson criteria (using three signs as the cutoff) for diagnosing severe disease is only 40% to 88% and the specificity is only 43% to 90%. The positive predictive value is approximately 50% and the

negative predictive value around 90%. Therefore, the best use of Ranson's score is to exclude severe disease.

CRITERIA FOR PANCREATITIS NOT DUE TO GALL STONES:

At admission or diagnosis:

Age more than55 years

WBC count > 16,000/mm^3

Blood sugar> 200 mg/dL

Serum LDH> 350 IU/L

AST > 250 U/dL

During initial 48 hours:

Fall in hematocrit> 10 percentage points

BUN elevation > 5 mg/dL

Serum calcium level < 8 mg/dL

Arterial Po_2 less than 60 mm Hg

Base deficit more than 4 meq/L

Estimated fluid sequestration > 6 L

CRITERIA FOR PANCREATITIS DUE TO GALL STONES

On admission or diagnosis:

Age > 70 yrs

WBC count > 18,000/mm^3

Blood sugar> 220 mg/dL

Serum LDH> 400 IU/L

AST > 250 U/dL

During initial 48 hours:

Fall in hematocrit greater than 10 percentage points

BUN elevation > 2 mg/dl

Serum ca^{2+} level < 8 mg/dl

Base deficit more than 5 meq/L

Estimated fluid sequestration > 4 L

APACHE-II Scores

APACHE-II is another commonly used scoring system in the United States to predict severity. It has the advantage of being able to be used on a daily basis and has similar positive and negative predictive values as the Ranson score at 48 hours after admission. The APACHE-II system assigns points for 12 physiologic variables, for age, and for chronic health status, in generating a total point score. The 12 variables are temperature; heart rate; respiratory rate; mean arterial blood pressure; oxygenation; arterial pH; serum potassium, sodium, and creatinine; hematocrit; white blood cell (WBC); and Glasgow Coma Scale. APACHE-II scores on admission and within 48 hours help distinguish mild from severe pancreatitis and to predict death. Most patients survive if APACHE-II scores are 9 or less during the first 48 hours. However, patients with APACHE-II scores of 13 or more have a high likelihood of dying. At admission, sensitivity is 34% to 70%, and specificity is 76% to

98%. At 48 hours, sensitivity remains less than 50%, but specificity is close to 90% to 100%. Strong drawbacks are its complexity, its low sensitivity on admission, and the fact that at 48 hours the score is no better than other scoring systems.Like the Ranson criteria, the APACHE-II score has its highest value in predicting mild disease.

BISAP

The problem with scoring systems is that they are cumbersome, using multiple variables. As described above, accuracy in predicting morbidity and/or mortality of the most commonly used scoring systems, Ranson and APACHE, is typically not achieved until 48 hours. By this time, it is usually apparent that the patient has developed severe disease manifested by organ failure. In order to develop a simple scoring system for patients with acute pancreatitis that would be useful within the first 12 hours from admission, the Pancreas Center at Brigham and Women's Hospital performed a series of studies retrospectively and prospectively. The studies were performed on a large database including almost 37,000 patients and more than 200 hospitals. After careful analysis, including a validation study, they determined that a simple system that included 5 variables could accurately determine severity early in the course of the disease. The scoring system, referred to as BISAP (Bedside Index for Severity in Acute Pancreatitis), also uses the first letter of each parameter

for 1 point. The BISAP score provides a single point for 5 parameters: blood urea nitrogen (BUN) greater than 25 mg/dL, impaired mental status, systemic inflammatory response syndrome, age greater than 60, and/or the presence of a pleural effusion, for a possible total of 5 points. A BISAP score greater than 3 is associated with a seven- to twelve-fold increase in developing organ failure. Accurate, yet much easier to use, this new simple scoring system appears to be useful in the early identification of patients who are at risk of developing complications and mortality.

The BISAP includes:

1) Blood urea nitrogen (BUN) >25 mg / dl.
2) Impaired mental status (GCS < 15).
3) SIRS.
4) Age >60 years.
5) Pleural effusion.

SIRS was defined by presence of two or more of the following criteria:

1) Pulse rate > 90/min.
2) Respiratory rate > 20/min or PaCO 2 < 32 mm Hg.
3) Temperature >100.4 F or < 96.8 F / < 36 or > 38 ° C.
4) WBC count >12,000 or < 4,000 cells/$mm^{3,}$ or presence of more than 10% immature blasts.

GLASGOW – IMRIE SCORE

Glasgow score is a slightly simplified list (eight criteria) that is used commonly in the United Kingdom. It has similar drawbacks to the Ranson score.

Within 48 hours

- WBC>15,000
- AST>300
- LDH>600
- Glucose>180
- BUN>45
- PaO2<60
- Calcium<2meq/l
- Albumin<3.2gms%

Advantage is easy to calculate and one need not wait for 48 hours

ORGAN FAILURE

There is considerable interest among pancreatologists in using organ failure to predict severity. The Atlanta criteria defined which organ systems are of importance: pulmonary, renal, and cardiovascular. However, these criteria did not attempt to quantitate or prognosticate using organ failure. It has been appreciated that multiorgan failure or persistent single organ failure has a greater associated mortality than

transient single organ failure. Multisystem organ failure is defined as two or more organs failing on the same day, rather than one organ failing on one day and another failing on the subsequent day. Patients with multisystem organ failure or persistent organ failure have a much higher mortality rate (approaching 50%) compared with patients with single and transient organ failure. Persistent organ failure is defined as lasting greater than 24 hours regardless of intervention. Survival among patients with organ failure at admission has also been shown to correlate with the duration of organ failure. When organ failure is corrected within 48 hours, mortality is close to zero. When organ failure persists for more than 48 hours, mortality is 36%. The Marshall Scoring System for organ failure is commonly used by intensivists for patients admitted to an intensive car unit. Data have not yet been generated using this system to prognosticate mortality in acute pancreatitis. Studies are needed to determine if this scoring system improves on the Ranson and APACHE scoring systems.

MODIFIED MULTIORGAN DYSFUNCTION SCORE

	Score				
Organ system involved	1	2	3	4	5
Cardiovascular					
PAHR (beats/min)	≤10	10–15	30–15	20–30	>30
Respiratory					
Pao_2/Fio_2 (mmHg)	>300	300–225	150–225	75–150	<75
Renal					
Creatinine (μmol/l)	<100	100–200	200–350	350–500	>500
Neurological					
Glasgow Coma Score	15	14–13	12–10	9–7	≤6
Haematological					
Platelet count ($\times 10^9$/l)	>120	80–120	50–80	20–50	≤20
Hepatic					
Bilirubin (μmol/l)	<20	20–60	60–120	120–240	>240

LABORATORY MARKERS

Because the degree of elevation of serum amylase and lipase does not distinguish mild from severe pancreatitis, other factors have been examined.

HEMATOCRIT

A high hematocrit on admission, or one that fails to decrease after 24 hours of rehydration is thought to be a sign of hemoconcentration due to retroperitoneal fluid loss and thus a marker of severe disease. One study showed that a hematocrit greater than 44% had a sensitivity of 72% on admission and of 94% after 24 hours in detecting organ failure. The negative predictive value at 24 hours was 96%. Although one study from Germany found no correlation between admission hematocrit and organ

failure, most investigators have found hematocrit to be important in the management of patients with acute pancreatitis. An elevated hematocrit (>44%) is a predictor for the development of necrosis. The hematocrit should be observed at admission for prognostic purposes and followed prospectively to assist in guiding the rate of intravenous hydration.

BLOOD UREA NITROGEN

Several prognostic scoring systems, including the Ranson criteria and BISAP, incorporate blood urea nitrogen (BUN) for the prediction of mortality in patients with acute pancreatitis. Hemoconcentration, as described above, has been shown to be an accurate predictor of necrosis and organ failure. Both BUN and the hematocrit or hemoglobin are routine laboratory tests that may provide information on changes in intravascular volume status. Either test may be used in monitoring the early response to initial fluid resuscitation. Wu and colleagues recently performed a large observational cohort study on data from 69 U.S. hospitals and found that BUN may be superior to hemoglobin (not hematocrit). For every 5 mg/dL increase in BUN during the first 24 hours, the age- and gender-adjusted odds ratio for mortality increased by 2.2. Of multiple routine laboratory tests examined, BUN yielded the highest accuracy at 24 hours and 48 hours. Although further study is needed, this paper suggests that following serial BUN measurements

would be the most valuable single routine laboratory test for predicting mortality in acute pancreatitis.

C-REACTIVE PROTEIN

CRP is an acute-phase reactant produced by the liver and is used extensively in Europe as a marker of severe pancreatitis. CRP is inexpensive to measure and readily available. The sensitivity for detecting severe disease is 60% to 100% (using cutoffs of 100 to 210 mg/L, or 10 to 21 mg/dL) and the specificity is 75% to 100%.

INTERLEUKIN-6

IL-6 is an acute-phase-reactant cytokine that is produced by a variety of cells and induces hepatic synthesis of CRP. Several studies have shown that it is a reasonably good marker to differentiate mild from severe disease, but the test is not readily available.

POLYMORPHONUCLEAR LEUKOCYTE ELASTASE

Polymorphonuclear leukocyte elastase rises very early (before CRP) in acute pancreatitis. High levels have been reported to differentiate severe from mild disease, but the test is not generally available.

PHOSPHOLIPASE A_2

PLA_2 is involved in the release of prostaglandins from cell membranes and degrades surfactant in the lung. It may play a role in the pulmonary dysfunction associated with acute pancreatitis. Levels of catalytic type II PLA_2 have been reported to differentiate between mild and severe disease within 24 hours of admission.

URINARY TRYPSINOGEN ACTIVATION PEPTIDE

Urinary TAP may serve as an early predictor of severity in patients with acute pancreatitis. Unlike other markers of severity, such as CRP, TAP is not a surrogate marker of inflammation. Normally trypsinogen is cleaved to trypsin in the intestinal lumen by the enzyme enterokinase. Premature intrapancreatic activation during acute pancreatitis results in the release of TAP. The degree of pancreatic necrosis and systemic inflammatory response or sepsis is directly related to TAP concentration. Elevated urinary TAP (>30 nmol/L) correlates with disease severity. The test can be applied within 12 hours of admission. The positive predictive value of an elevated TAP is 80% and the negative predictive value approaches 100%.

PROCALCITONIN

This propeptide is another acute-phase reactant that has been shown to differentiate mild from severe acute pancreatitis within the first 24 hours after symptom onset. A serum strip test has been developed that has a sensitivity of 86% and a specificity of 95% in detecting organ failure.

CHEST RADIOGRAPHY

A pleural effusion documented within 72 hours of admission by chest radiography (or CT) correlates with severe disease.

COMPLICATIONS OF ACUTE PANCREATITIS

LOCAL COMPLICATIONS

- Pancreatic Necrosis
    - Sterile
    - Infected - abscess
- Pancreatic Pseudocyst
- Pancreatic Ascites
- Intraperitoneal hemorrhage
- Splenic vein Thrombosis
- Colonic stricture

- Obstructive jaundice
- Pancreatic pseudo aneurysm

SYSTEMIC COMPLICATIONS

- *Pulmonary*
    - Pleural effusions
    - Atelectasis
    - Pneumonitis
    - Mediastinal abscess
    - ARDS , Respiratory failure
- *Cardiovascular*
    - Shock- Hypovolaemic & septic
    - Pericardial effusion
- *Hematologic*
    - DIC
- *Gastrointestinal*
    - PUD
    - Erosive gastritis
    - Erosion of Gastroduodenal vessels or inferior and superior arteries
    - Portal vein thrombosis

- *Renal*
    - Oliguria
    - Azotemia
    - Renal artery/vein throbosis
    - ATN, Acute renal failure
- *Metabolic*
    - Hypocalcemia
    - Hyperglycemia
    - Encephalopathy
    - Sudden blindness (Purtscher's retinopathy)
- *Central nervous system*
    - Psychosis , Fat emboli
- *Miscellaneous*
    - Septicemia
    - Subcutaneous fat
    - Necrosis
- **LONG TERM COMPLICATIONS**
    - Chronic Pancreatitis
    - Abdominal Pain
    - Steatorrhea

- Exocrine insufficiency (pancreas has a 90% reserve for the secretion of digestive enzymes)
- DM, i.e.Endocrine Insufficiency
- Pseudocyst

TREATMENT

The mainstay of treatment of acute pancreatitis is elimination of inciting cause whereas treatment of chronic pancreatitis mainly involves long term management of pain, pancreatic exocrine & endocrine deficiency. The treatment needs to be tailored to each individual patient, considering the techniques available in each Institution.

General principle includes correcting any underlying predisposing factors: Early ERCP in patients with gallstone pancreatitis who have obstructive jaundice or biliary sepsis, Reversal of hypercalcemia, Cessation of possible causative drug, The administration of insulin to the poorly controlled diabetic with marked hypertriglyceridemia and lastly the pancreatic inflammation itself.

Basic management of pancreatitis include

- Estimating severity
- Fluid & electrolyte management

- The Control of Pain
- The Control of Nausea, Vomiting, Ileus
- Nutritional support
- Limiting progression

IMMEDIATE ASSESSMENT

Clinical assessment includes great care to assess respiratory, cardiovascular and renal compromise. Body mass index: There is considerable risk (> 30 kg/m2) or much greater risk > 40 kg/m2. Chest X-ray: Is there a pleural effusion / pulmonary edema / ARDS present? Contrast-enhanced CT: Is there more than 30% of the volume of the pancreas malperfused. The use of various scoring systems to predict severity of the disease and presence of organ failure.

Patients with predicted severe acute pancreatitis should be treated in intensive care unit. In severe pancreatitis, ICU monitoring and support of pulmonary, renal, circulatory, and hepatobiliary function may minimize systemic sequelae.These patients need monitoring of vital signs and urine output every few hours in the first 24 to 48 hours with further ongoing monitoring for other complications that might arise .

***FLUID AND ELECTROLYTE MANAGEMENT*:**

Transudation of fluid from the intravascular space to the peritoneal cavity is the principle cause of hypovolemia. “Cytokine Storm” causes leaky capillaries and results in transudation. Other important cause are vomiting, Insensible fluid loss due to tachypnoea and fever.Vomiting usually leads to chloride and H+ loss leading to hypochloremic metabolic alkalosis.

Fluid resuscitation is particularly important because patients with necrotizing pancreatitis accumulate vast amounts of fluid in the injured pancreatic bed.Inadequate hydration can lead to hypotension and acute tubular necrosis.Fluid depletion damages pancreatic microcirculation and results in pancreatic necrosis.Approximately 250 to 300 cc of intravenous fluids per hour are typically required for 48 hours if the cardiac status permits.

Adequate fluid replacement can be assessed by improvement in vital signs and urine output and reduction in hematocrit over 24 hours, particularly if it was high at the onset In some patients, a low urine output may already reflect the development of ATN rather than persistent volume depletion. Assessment of the patient’s volume status should be

determined by heart rate, blood pressure, urine output and jugular venous pressure.

Fluid management may be difficult when hypovolemia is combined with ARDS.Fluid balance flow sheet is useful in such cases.Pulse, BP & urine output are unreliable for fluid requirement in this setting. Hematocrit may be useful in determining the fluid requirement. Measurement of CVP will be accurate in determining fluid requirement in this setting.

Potassium chloride should be added to the intravenous fluids to achieve 100 mEq/day. Glucose levels greater than 13.9 mmol/L (250 mg/dL) necessitate insulin administration. A blood transfusion is indicated if the patient's hematocrit is less than 25%; values ranging from 30 to 35% are considered optimal for pancreatic parenchymal perfusion.

Hypoalbuminemia may be due to many patients are chronic alcoholics Due to ongoing losses Albumin levels further depressed as fluid losses are treated by albumin free colloids Treat it with albumin infusion, if levels are very low.

Hypocalcemia is common, particularly during an acute attack, usually attributable due to low serum albumin. No treatment required, if ionized

calcium levels are normal. Aggressive calcium repletion required if hypocalcemic features like tetany, carpopedal spasm occur.

Oxygen saturation needs to be assessed routinely and supplemental oxygen administered to maintain arterial oxygen saturation of greater than 95 percent. Blood gas analysis should be done if SaO2 is less than 95 percent or if clinical situation demands. Prophylaxis against DVT should be considered in bedridden patients. Intermittent pneumatic compression may be the preferred method because of the theoretical risk of precipitating pancreatic hemorrhage with anticoagulation.

CONTROL OF PAIN

Pain of pancreatitis may be very severe and difficult to control. Severe pain should be treated with meperidine *50 to 100 mg IM q 3 to 4 h* in patients with normal renal function. Morphine causes the sphincter of Oddi to contract and should be avoided.

Nausea & vomiting can result in significant fluid & electrolyte loss. Furthermore, retching can lead to UGI bleeding (mallory-Weiss syndrome). Although institution of NG tube drainage has not been shown to alter eventual outcome, to increase patient comfort NG tube drainage can be done. Moreover instillation of NG tube dramatically reduces pain in many patients of acute pancreatitis.

NUTRITIONAL SUPPORT

Patients with severe pancreatitis may not be able to eat for prolonged periods. Traditionally these patients have been given Total Parenteral Nutrition. Recent studies show most patients of pancreatitis, even those with severe pancreatitis, can tolerate small amounts of enterally administered nutrients (Through NG or NJ tube).

SIRS, sepsis, organ failure, and ICU stay were globally improved in the enterally-fed patients. The acute phase response and disease severity scores CRP, APACHE II) were significantly improved following enteral nutrition without any change in the CT scan scores. Enteral feeding modulates the inflammatory and sepsis response in acute pancreatitis and is clinically beneficial. There is also no doubt that probiotics associated with enteral feeding may become an alternative therapy replacing early antibiotic use to prevent infection in severe pancreatitis.

To limit the progression of disease the patient should be advised to stop smoking to stop smoking, to abstain from alcohol and to stop the causative drugs.

ANTIBIOTICS IN PANCREATITIS

Infectious complications are still regarded as the primary cause of mortality in severe pancreatitis. It is essential to identify the presence of pancreatic necrosis and take measures to prevent infection. The current recommendation is the use of a systemic antibiotic such as imipenem-cilastatin 500 mg three times a day for 2 weeks in patients with documented pancreatic necrosis. An acceptable strategy would be to perform a CT scan with intravenous contrast at days 4–7 and begin imipenem if necrosis is present. The use of early antibiotic treatment with imipenem has been shown to decrease the need for surgical intervention. If there is clinical evidence of infection, pancreatic necrosis should be sampled by CT-guided fine needle aspiration (FNA). If infection is confirmed, the tissue should be treated by surgical debridement, either via open access or percutaneously.

Prophylaxis is indicated in only severe cases, not useful for mild cases. Antibiotic prophylaxis significantly reduced sepsis by 21.1% and mortality by 12.3% compared with no prophylaxis. There was also a non-significant trend toward a decrease in local pancreatic infections. Antibiotic prophylaxis decreases sepsis and mortality in patients with acute necrotizing pancreatitis. All patients with acute necrotizing

pancreatitis should receive prophylaxis with an antibiotic of proven efficacy.

Few studies suggest prophylactic antibiotics have no benefit in the outcome of an episode of acute pancreatitis. It favours emergence of resistant strains at the site of pancreatic injury. Recently fungal strains have been identified in the infected pancreatic necrosis cases**.**

ROLE OF SURGERY

It is to treat the underlying cause and for complications. Patients with mild pancreatitis due to gall stones should undergo cholecystectomy in the same admission.

Some have argued that a lack of stabilization or improvement with full supportive intensive care therapy over 72 h should constitute an indication for surgical intervention to establish intra-abdominal peritoneal lavage, but no randomized study has validated this approach. When a patient has clinical evidence of sepsis (usually > 7 days of onset) unexplained by normal microbiology studies a CT scan should be performed and FNA with immediate Gram stain and subsequent culture of the fluid.

Traditionally, an anterior open surgical approach either through a transverse upper abdominal or a vertical incision has been routinely advocated with exploration of the area of necrosis and infection, using digital dissection or gentle instrumentation, to remove the dead tissue.

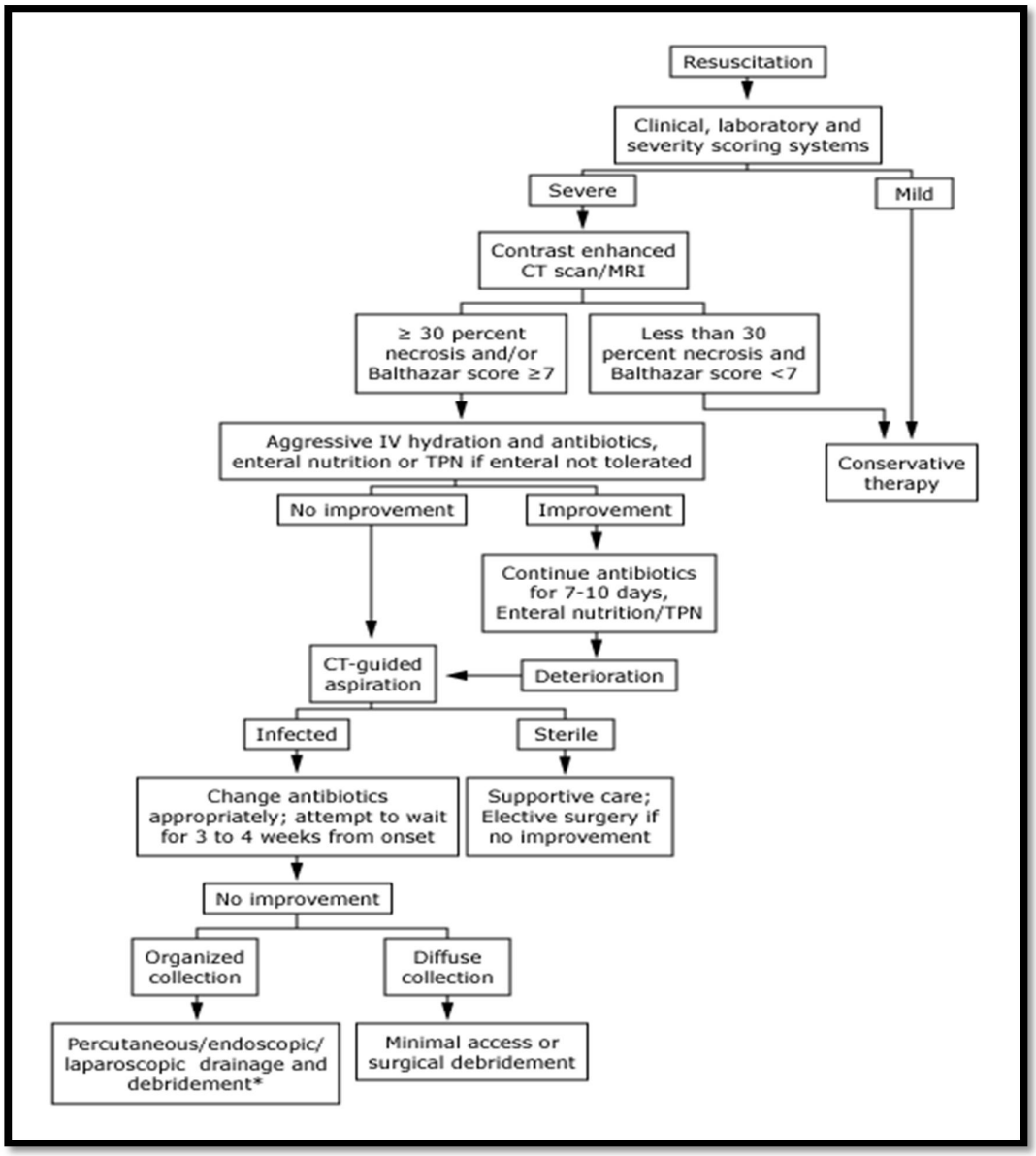
Postoperatively, lavage through strategically placed drains should continue at a rate of 1–2 L/day and this may be required for 3 to 4 weeks with a 30% chance of a repeat operation being necessary because of recurrent sepsis.

Where venous bleeding and oozing of blood is particularly troublesome packing of the upper abdomen with large cotton packs enclosed in paraffin gauze or a similar non-adherent material may be necessary. Alternatives to open surgery that are being actively investigated include both anterior laparoscopic and retroperitoneal percutaneous approaches. As one of the major purposes of surgical therapy in severe AP relates to minimizing the risk of a further episode of pancreatitis it is logical and wise to remove the gallbladder and check for any residual stones in the CBD at the same operative procedure.

TREATMENT OF UNPROVEN VALUE

Peritoneal dialysis (designed to eliminate proinflammatory factors released into abdomen) is of no benefit. Drugs reducing gastrointestinal

or pancreatic secretions like atropine, antacids, somatostatin analogues (octreotide, calcitonin) have not been beneficial. Inhibitors of proteolytic enzyme (aprotinin, gabexate) are useless unless begun before the onset of pancreatitis. Hypothermia, thoracic duct drainage, plasmapheresis are still in experimental stage.



INFLIXIMAB IN ACUTE PANCREATITIS

In acute edematous pancreatitis and in severe necrotizing pancreatitis, the drug significantly decreased serum amylase activity and the histopathologic score. In severe necrotizing pancreatitis, it ameliorated both parenchymal and fatty tissue necrosis of the pancreas. It also alleviated alveolar edema and ARDS-like pulmonary complications, but this difference was not significant.

RESVERATROL IN ACUTE PANCREATITIS

To evaluate the protective and antioxidative effect of resveratrol, a stilbene derivative, in acute pancreatitis induced by tert-butyl hydroperoxide injection. Changes in pancreas were much less pronounced in the rats which received resveratrol for 8 days prior to tert-butyl hydroperoxide injection.In this way it seems that stilbene derivatives may prevent pancreatic cells from undergoing structural changes during acute pancreatitis experimentally induced in rats

AIMS AND OBJECTIVES OF THE STUDY

- To compare CT severity scoring system with the multifactorial scoring systems: Glasgow/IMRIE and BISAP in predicting severity, pancreatic necrosis and mortality in a prospective cohort of patients with acute pancreatitis which is analysed retrospectively
- To find out which among the three studies has strong association in predicting the complications?
- To stratify the patients based on the predicted severity and provide intensive care treatment earlier in the course of disease and thereby decrease morbidity and mortality.

MATERIALS AND METHODS

SETTING:

Department of general surgery, Stanley medical college and hospital, Chennai. The study was done after obtaining institutional ethical committee approval.

DURATION:

Nov 2012 to Nov 2013

STUDY DESIGN:

Observational comparative analytical study

INCLUSION CRITERIA:

- Age >15 years including both sexes.
- Patients with clinical features of pancreatitis and S. amylase/ S. lipase equal to or more than 3 times the upper limit of normal or
- Radiological evidence of presence of acute pancreatitis.

EXCLUSION CRITERIA:

- Patient less than 15 yrs in age
- Proven cases of chronic pancreatitis.
- Hereditary pancreatitis.
- Patients with comorbidities like COPD, renal impairment, immunosuppressive state, etc.

METHODOLOGY

Demographic, clinical, and laboratory data of all consecutive patients with a primary diagnosis of ACUTE PANCREATITIS during a one year period is prospectively collected for this study. A retrospective analysis of the abdominal CT data is performed. CT severity index as well as two clinical scoring systems: Glasgow criteria / IMRIE'S prognostic criteria and Bedside Index for Severity in Acute pancreatitis (BISAP) were comparatively evaluated with regard to their ability to predict the severity of acute pancreatitis on admission (within 48 h of hospitalization).

Clinically severe Acute was defined as one or more of the following: mortality, temporary/ persistent organ failure and/or the presence of local pancreatic complications that require intervention. All CT scans were reviewed in consensus by two radiologists, each blinded to patient outcome. The accuracy of each imaging and clinical scoring system for predicting the severity of AP was assessed using appropriate statistical tools.

First 50 patients attending the surgical emergency ward with clinical features of Acute Pancreatitis are evaluated clinically and subjected to laboratory and radiological investigations as per the designed proforma. (annexure 1). Data pertinent to the scoring systems will be

recorded within 24hr of admission to the hospital. Once diagnosis is established the patient disease severity will be assessed by following the scoring systems

- CT SEVERITY INDEX
- MODIFIED GLASGOW
- BISAP

The etiology of the disease has been analysed and divided into alcoholic, biliary, drug induced, hypertryglyceridimia and idiopathic. Biliary pancreatitis has been confirmed only after visualization of stone in gall bladder or biliary tract with the help of an ultrasound or computed tomography.

The outcome of the patient is recorded in the form of survivor or non- survivor. Complications that developed in the course of disease while the patient was in the hospital have been recorded. All patients have been followed up until discharge or death.

OBSERVATION AND RESULTS

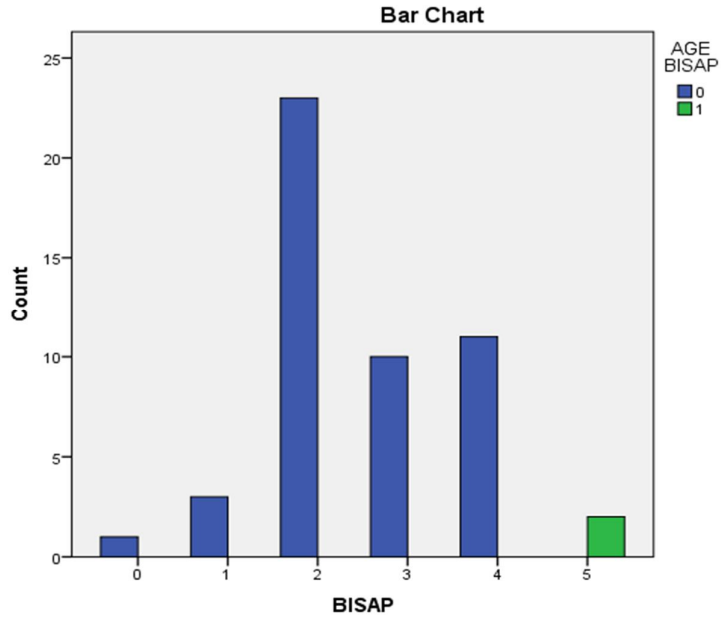
The study was conducted in general surgery department, Stanley medical college for a period of one year. Totally 50 patients with features of acute pancreatitis were enrolled in the study after obtaining proper informed consent.

TABLE 5: AGE DISTRIBUTION

AGE IN YEARS	NO. OF PATIENTS	PERCENTAGE
21-30	13	26%
31-40	15	30%
41-50	12	24%
51-60	9	18%
61 and Above	1	2%
TOTAL	50	100%

Among the 50 cases nearly 80% are between the age group of 21 to 50 years and only 2% of the people above 60 years had the disease.

GRAPH 1: BISAP VS AGE



The age cut off in BISAP score is 60 years and for patients above the age of sixty, the severity is predicted to be more and hence one point is added. In the above study only two patients were above 60 and the graph shows that both the patients had maximum BISAP score suggestive of severe pancreatitis.

GRAPH 2: GLASGOW VS AGE

In Glasgow the cut- off age is 55 and arbitrarily patients more than 55 years have more severe pancreatitis than patients less than 55 yrs but the above graph in my study showed that among the 5 patients more than 55 yrs two had severe and three had mild pancreatitis according to Glasgow score

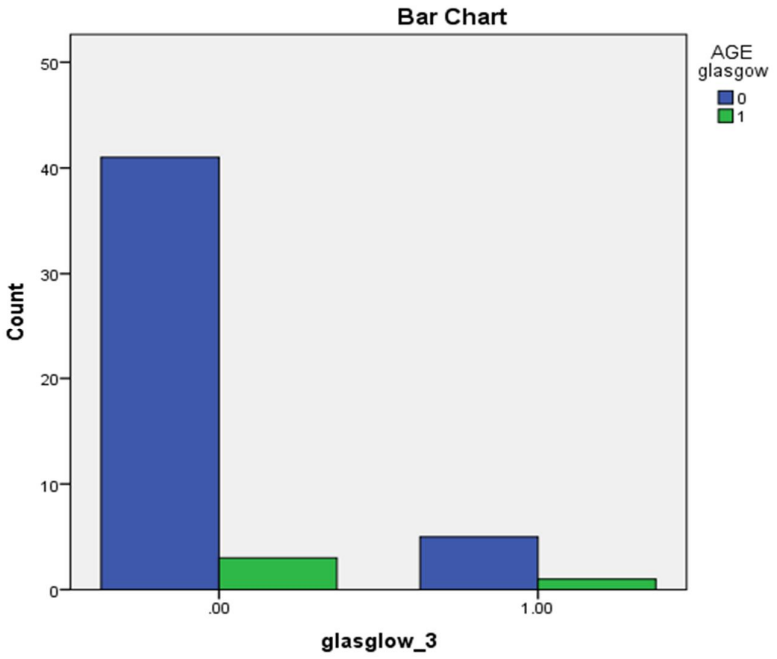
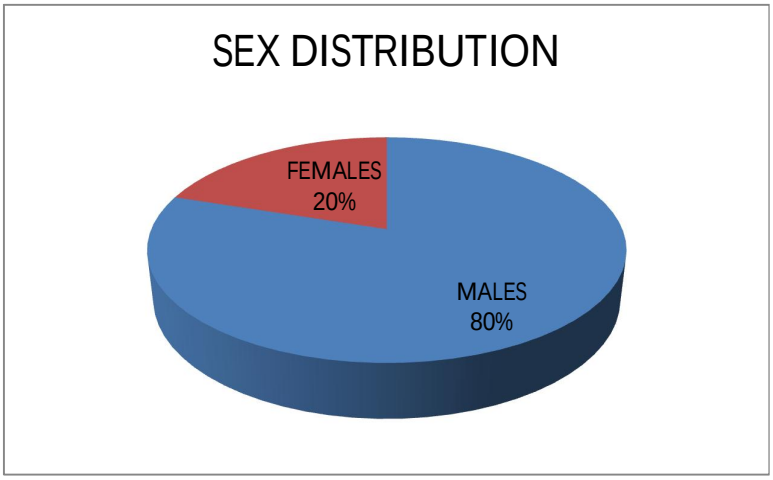


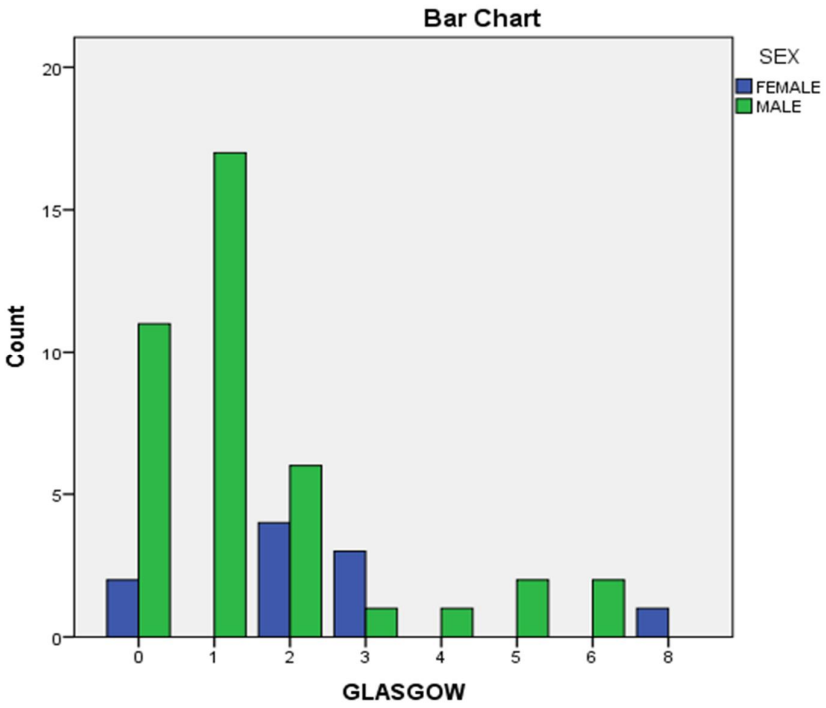
TABLE 6: SEX DISTRIBUTION

AGE IN YRS	NO. OF MALES	NO. OF FEMALES	PERCENTAGE	
			males	Females
21 - 30	12	1	24%	2%
31 – 40	10	5	20%	10%
41 – 50	10	2	20%	4%
51 – 60	8	1	16%	2%
>60	0	1	0	2%
TOTAL	40	10	80%	20%



80% of the cases are male and among males only 16% are above 50 years of age. Among females which form the remaining 20% only 4% are above 50 yrs of age.

GRAPH 3: SEX DISTRIBUTION IN GLASGOW SCORE



The above graph compares the variation of Glasgow score between both sexes and it shows males sex is predominantly affected by mild pancreatitis (35 male patients have score equal to or below three) and similarly females are also mostly experiencing mild pancreatitis (only one female out of ten had a score more than three).

SEX DISTRIBUTION IN BISAP SCORE

In the below graph, we compare variations of bisap score with sex. Nearly 45% of males and 40% of females are having a score of 3 or more

as against Glasgow which shows only 12.5% of males and 10%of females with score more than three.

GRAPH 4: BISAP SCORE VARIATION IN BOTH SEX

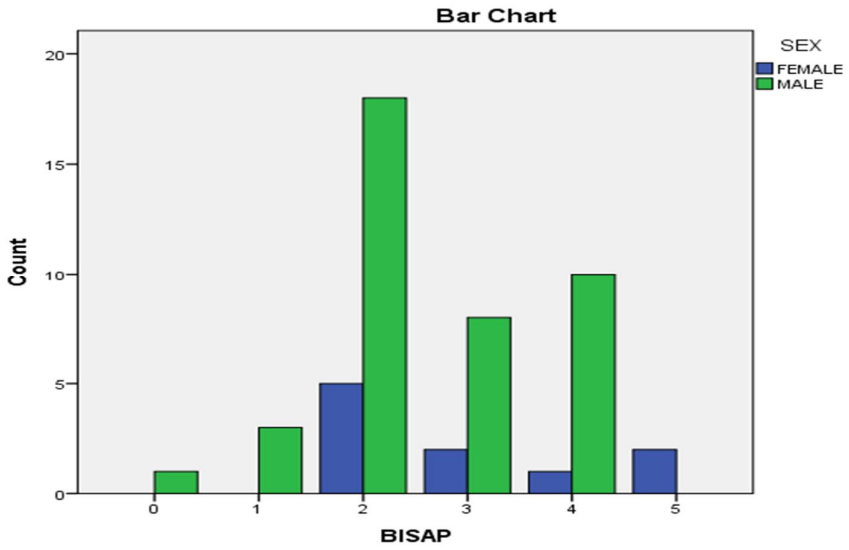
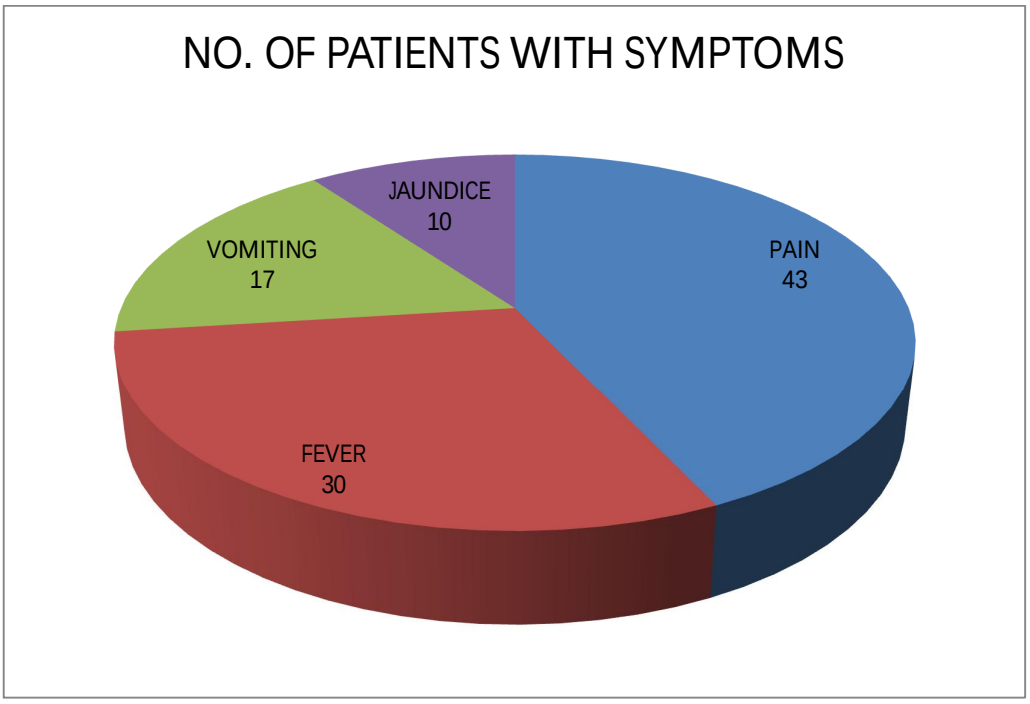


TABLE 7: CLINICAL FEATURES

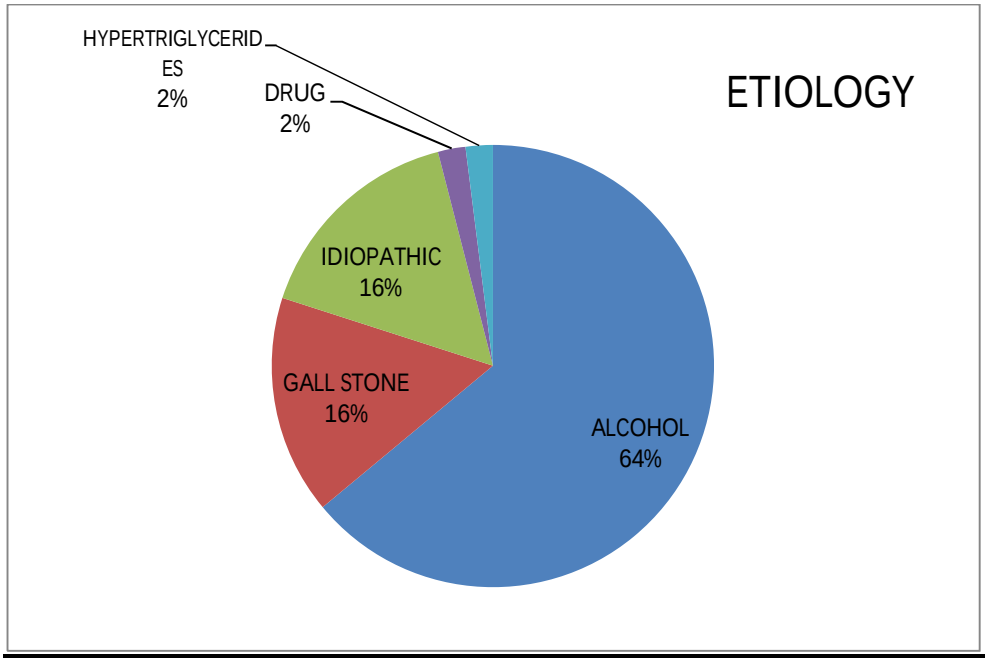
Symptoms	No. of patients	Percentage (%)
Pain abdomen	43	**86**
Fever	30	**60**
Vomiting	17	**34**
Jaundice	**10**	**20**



At the time of admission nearly 85% had abdominal pain with or without other features as their presenting complaint. Remaining 14% had either vomiting, jaundice or fever as their complaints.

TABLE 8: ETIOLOGIES

Etiology	No. of patients	Percentage (%)
Alcohol	32	**64**
Gall stone disease	8	**16**
Drug induced	1	**2**
Hypertriglyceridemia	1	**2**
Trauma	Nil	**0**
Idiopathic	**8**	**16**



Among the 50 cases nearly 64% are predicted to have alcohol as their etiology with gall stone disease forming 16% and pancreatitis due idiopathic causes forming another 16%.

TABLE 9: NO. OF PATIENTS WITH COMPLICATIONS

Complication	No. of patients	Percentage (%)
Acute renal failure	3	18.7
Acute respiratory distress syndrome	1	6.25
Pancreatic necrosis	6	37.5
Multi-organ dysfunction syndrome	4	25
Septicemia	2	12.5
Pseudo cyst	4	25
Hypocalcemia	1	6.25

Out of 16 patients who had either organ failure or local complications or both, acute renal failure was seen in 18% and MODS was seen in 25% of patients. Local complications like pancreatic necrosis were seen in 37.5% cases and pancreatic psuedocyst in 25% cases.

TABLE 10: COMPARING SCORING SYSTEM WITH ORGAN FAILURE

SCORING SYSTEM		NO. OF PTS	ORGAN FAILURE	PANCREATIC COMPLICATIONS	MORTALITY
BISAP	<2	27	NIL	NIL	NIL
	≥ 3	23	9	10	4
GLASGOW	<2	40	3	5	2
	≥3	10	6	5	2
CTSI	<3	44	6	4	1
	≥4	6	3	6	3

Out of 50 cases 16 developed organ failure or local pancreatic complications or both with 4 cases of mortality among them.

BISAP score was able to predict both organ failure and local complications when the cut off score is 2 (that is all patients with score 3

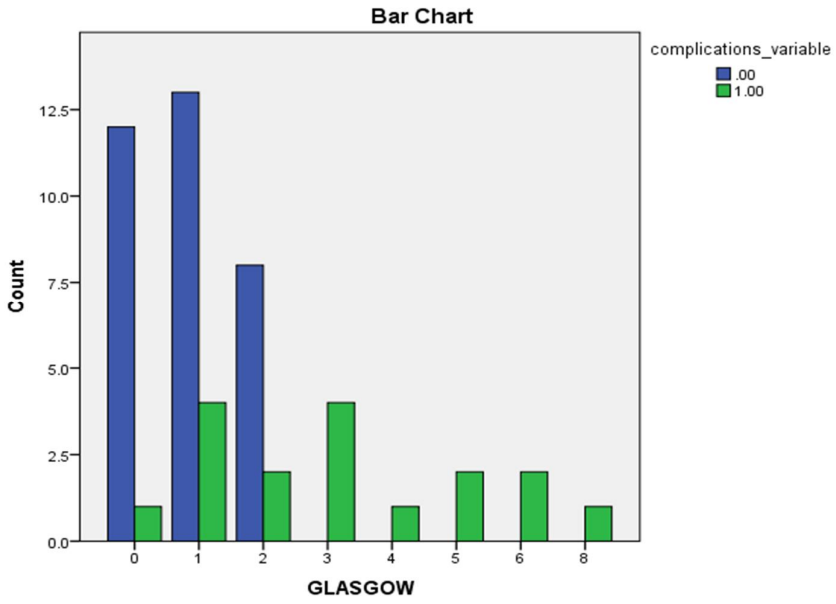
or more had either organ failure or local complications). All the four cases of mortality had score 3 or more. In other words severity and mortality prediction when the cut off value is 2 is 100%.

Glasgow score was able to predict organ failure in 66% of cases when score was at or above 3 and only 50% of local pancreatic complications and 50% of mortality.

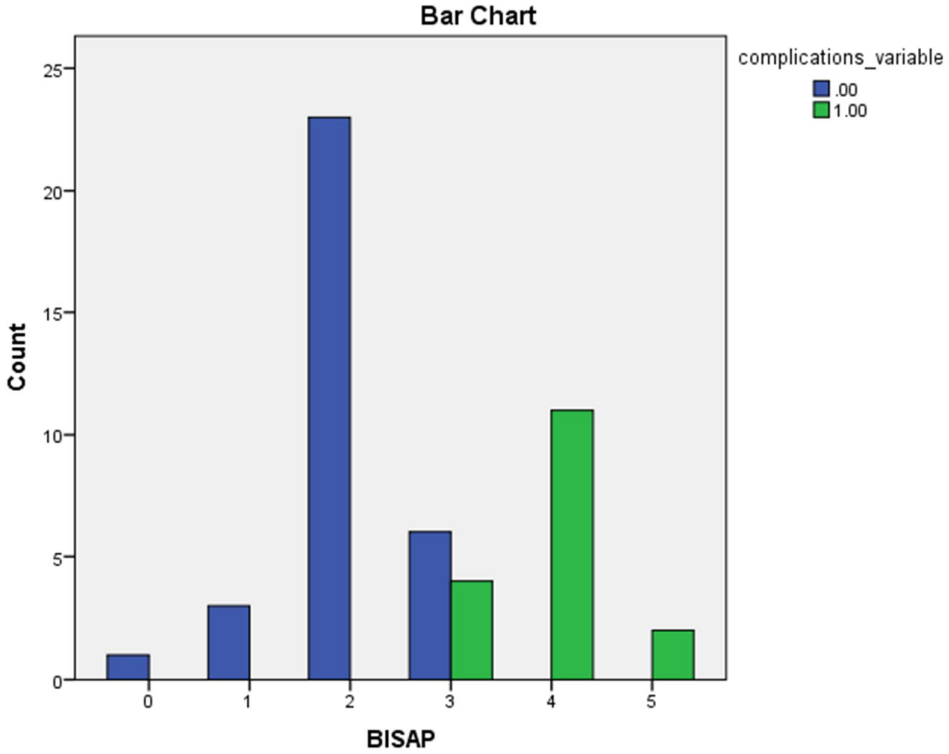
In CTSI when an arbitrary cut off value of 4 (that is patients with necrosis) is set only three patients above 4 developed organ failure and three patients had mortality. Hence when necrosis is indicated in CTSI organ failure is only 33% and mortality is 50%.

GRAPH 5: GLASGOW VS COMPLICATIONS

In this graph it can be seen that the number of people with Glasgow below two who have developed complications (either organ failure or local complications) is significant. This means nearly 37% of patient who were predicted to have mild pancreatitis developed complications.

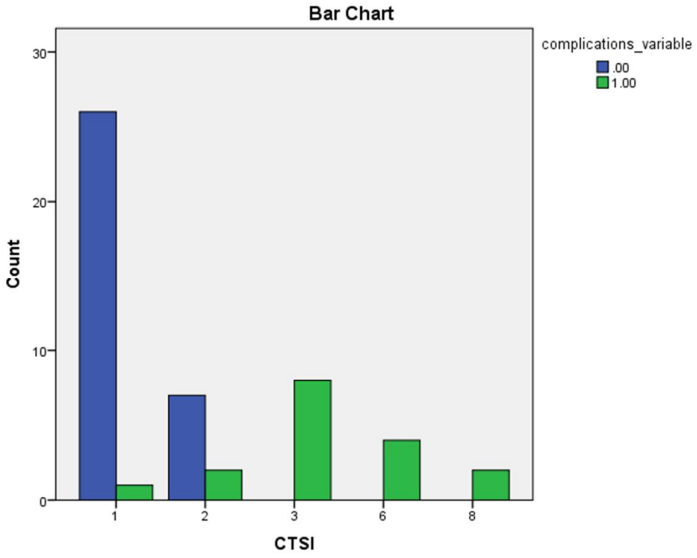


GRAPH 6: BISAP VS COMPLICATIONS



In the above graph, it is clearly indicated that no complications were seen when score is less than 2 and with score above 3 or more the complication pick up rate is 100%. It can also be seen that out of 23 patients who were predicted to have severe disease only 16 (that is 69%) developed complication.

GRAPH 7: CTSI VS COMPLICATION



In this graph it is seen that among 44 patients with score less than four (that is only pancreatic inflammation) 6 have developed either organ failure and hence 13% of complications has been missed in this complications.

TEST TO INDICATE THE CORRELATION OF THE RESULTS WITH THE PRESENCE OF COMPLICATIONS

H0: there is no correlation between the test results and the presence of complication

H1: There exist a significant correlation between the results of the test and the complication

		complications_variable
glasglow_3	Pearson Correlation	.514**
	Sig. (2-tailed)	.000
	N	50
BISAP_3	Pearson Correlation	.826**
	Sig. (2-tailed)	.000
	N	50
CTSI_4	Pearson Correlation	.514**
	Sig. (2-tailed)	.000
	N	50

Interpretation

Since the sig.(2 tail) p value is less than 0.05, there is a correlation between the test results and the presence of complication. The correlation analysis of the presence of complication with the three scoring methods reveals that the BISAP has high correlation of 82.6% than other two tests. Thus presence of complication has 82.6% effect on given positive results in the test. Glasgow and CTSI has 51.4% correlation.

TEST TO DETERMINE THE STRENGTH OF THE METHOD ON DETERMINING THE COMPLICATION (CRAMER'S V TEST)

BISAP

Crosstab

			complications_variable		Total
			.00	1.00	
BISAP_3	.00	Count	33	4	37
		% within complications_variable	100.0%	23.5%	74.0%
	1.00	Count	0	13	13
		% within complications_variable	0.0%	76.5%	26.0%
Total		Count	33	17	50
		% within complications_variable	100.0%	100.0%	100.0 %

H0: There is no association with the test results and the complication

H1: There is a significant association with the test results and the complication

Symmetric Measures

		Value	Approx. Sig.
Nominal by Nominal	Phi	.826	.000
	Cramer's V	.826	.000
N of Valid Cases		50	

Interpretation

The first table explains that in 23.5% cases the BISAP is negative when the subject actually has the complication. The Cramer's V test shows that there is a significant association of the test result to the presence of complication and the strength of association is 0.826 (i.e.) very high association.

GLASGOW

Crosstab

			complications_variable		Total
			.00	1.00	
glasglow_3	.00	Count	33	11	44
		% within complications_variable	100.0%	64.7%	88.0%
	1.00	Count	0	6	6
		% within complications_variable	0.0%	35.3%	12.0%
Total		Count	33	17	50
		% within complications_variable	100.0%	100.0%	100.0 %

H0: There is no association with the test results and the complication
H1: There is a significant association with the test results and the complication

Symmetric Measures

		Value	Approx. Sig.
Nominal by Nominal	Phi	.514	.000
	Cramer's V	.514	.000
N of Valid Cases		50	

Interpretation

The first table indicates that there is a 64.7% that the test would be normal when the subject is actually suffering from a complication. The strength of association is 0.514 which is moderate association.

CTSI

Crosstab

			complications_variable		Total
			.00	1.00	
CTSI_4	.00	Count	33	11	44
		% within complications_variable	100.0%	64.7%	88.0%
	1.00	Count	0	6	6
		% within complications_variable	0.0%	35.3%	12.0%
Total		Count	33	17	50
		% within complications_variable	100.0%	100.0%	100.0 %

H0: There is no association with the test results and the complication

H1: There is a significant association with the test results and the complication

Symmetric Measures

		Value	Approx. Sig.
Nominal by Nominal	Phi	.514	.000
	Cramer's V	.514	.000
N of Valid Cases		50	

Interpretation

The first table indicates that there is a 64.7% that the CTSI would be normal when the subject is actually suffering from a complication. The strength of association is 0.514 which is moderate association.

Note on Cramer's V test

0-0.30 → Weak association

0.31-0.70→ Medium or moderate association

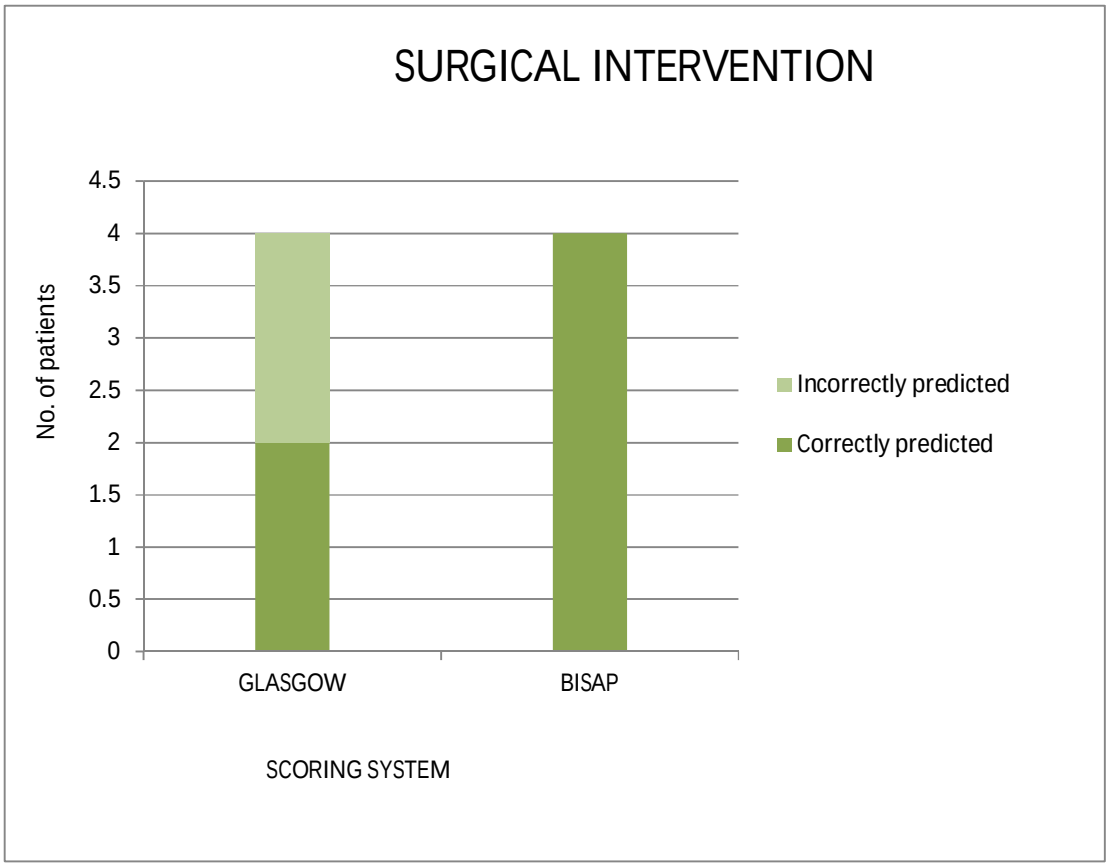
0.71- 1.0 → strong association

MANAGEMENT

All except four patients managed conservatively.

Three patients underwent emergency laparotomy. Two patient had acute severe necrotizing pancreatitis which was initially managed conservatively later they developed severe pancreatitis with intra-abdominal abscess, that required laparotomy and drainage procedure.

GRAPH 8 : PATIENTS REQUIRING SURGERY AS PER BISAP AND GLASGOW



One patient with severe disease have developed pseudocyst, underwent laparotomy and internal drainage.

Another patient who had severe pancreatitis underwent necrosectomy initially& has developed pancreatic fistula later, which was managed by pancreatic duct stenting. The BISAP score has predicted all the four patients correctly as severe pancreatitis, whereas GLASGOW score predicted only two of the cases as severe pancreatitis.

DISCUSSION

Acute pancreatitis is a disease with a large and varies spectrum of presentation. In acute pancreatitis with chances of high mortality, early hospitalization is needed to identify those who require aggressive interventions to prevent the severe attack of AP.

In this study, the three different scoring systems (BISAP , GLASGOW and CTSI) were compared and analyzed to assess the severity in patients with acute pancreatitis. An attempt also made to compare this study with previous similar studies done by others.

Acute pancreatitis found to be 4 times more common in males than females in this study because alcohol consumption was the predominant cause which was more common in males. This result matches with previous study results, Vikesh K. Singh et al[38] (6:1), Papachristou et al[1] (5.1:1).

Patients less than 15 years of age were excluded in this study, because the normal values of heart rate and respiratory rate are higher at younger age group. So, if these patients had been included in this study, they could have got higher scores incorrectly and could have predicted incorrectly as at risk for developing severe pancreatitis, even with mild disease.

Age is considered as a significant contributory factor in predicting the outcome of severe acute pancreatitis. In my study BISAP scoring with age cut off as 60, all patients above this age had severe pancreatitis but in GLASGOW only 40% above the cut off age of 55 had severe pancreatitis and henceforth BISAP has better prediction than GLASGOW in old age patients. This variable cannot be applied to CTSI.

The most common etiological factor in this study was alcohol (64%) and matches with Bidarkundi et al[43](46.67%), but didn't correlate with results of Vikesh K. Singh et al[38] (21.4%), Papachristou et al[1] (14%) wherein gall stone disease found to be the most common cause, 27% & 36% respectively.

The most common presentation was predominantly abdominal pain (86%), followed by fever (60%), vomiting (34%) & other manifestations.

In this study, 34 patients were diagnosed to have mild acute pancreatitis and 16 patients found to have severe acute pancreatitis. All the 16 patients were correctly predicted by BISAP Score. The severity was assessed by correlating the scores with three factors: organ failure, necrosis and mortality.

The other two scoring systems namely GLASGOW and CTSI were able to pick up only 50% of complication rates.

On keeping the cut of value for BISAP as 3, GLASGOW as 3 AND CTSI as 4 and analyzing using PEARSON CORRELATION it was found BISAP had 82.6% correlation compared to GLASGOW and CTSI which only had 51.4% correlation. If BISAP score predicts the disease to be severe then there is 82% positivity that the patient will have acute severe pancreatitis.

In CRAMER V test the strength of association was found to be 0.826 for BISAP score which is very high for predicting complications. In other words only 23.6% of people with negative BISAP score will have complication. The strength of association for Glasgow and CTSI was 0.514 which is moderate association and there is 64.7% chance of negative score even the patient has severe complications. In this study, 4% underwent surgical intervention which comparable with Sarath et al.

In this study, 4 patients with severe acute pancreatitis were expired. All four deaths were correctly predicted by BISAP score. Three patients were expired due to MODS and one patient expired due to septicemia

In this study, 37.5% developed acute pancreatic necrosis, 25% developed MODS, 25% developed psuedocyst and 18% developed acute renal failure. All these complications were correctly predicted in patients with BISAP ≥ 3 and hence concluded that these are the patients in high

risk group, who requires intensive monitoring and probably early intervention if necessary.

BISAP score was found to have better correlation, high strength of association and diagnostic accuracy with less negative value, compared to GLASGOW and CTSI in predicting the severity of acute pancreatitis. Hence, BISAP score found to predict more number of patients, likelihood of progressing to severe disease. Larven et al stated in their study that, a prognostic scoring assay should preferably have high positive and negative predictive values or high negative predictive value to assess the severity of acute pancreatitis. Hence, BISAP is considered as better score in assessing the severity than GLASGOW and CTSI Score.

LIMITATIONS OF THIS STUDY ARE:

- Small number of patients in this study.
- The etiology in this study were found to be different from worldwide accepted one, hence might not be correct to compare with other studies.
- The GCS score used to assess the mental status of the patient got admitted were subject to interobserver variation.
- Various factors associated with the disease like cholangitis, alcohol withdrawal may interfere with the assessment of physiological scores, which may leads to difference in the results.
- Recently, it has been suggested that severe acute pancreatitis may have variable disease progression; therefore the lack of predictability might be associated with this disease variability.
- Variation in timing of presentation of patients to the hospital after onset of symptoms may interfere with assessment of the scoring systems.

CONCLUSION AND SUMMARY

- From this study, Alcohol (64%) was found to be the most common etiological factor for acute pancreatitis.
- Males were most commonly affected than female with a ratio of 4:1.
- The most common age groups of patients affected were in 2^{ND} to 4^{th} decade of life.
- The overall mortality in patients with severe acute pancreatitis was 8%.
- All the three scores were significant in predicting severity and complications but BISAP had the highest strength of association among the three to correctly predict severity and mortality.
- From this study, we conclude that the BISAP score could be a simple and better clinical scoring system for the evaluation of disease severity in acute pancreatitis than GLASGOW and CT severity index.

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INSTITUTIONAL ETHICAL COMMITTEE, STANLEY MEDICAL COLLEGE, CHENNAI-1

Title of the Work : A comparative evaluation of radiologic and clinical Scoring systems in early prediction of severity in acute pancreatitis

Principal Investigator : Dr.S.Vijay Raj

Designation : PG in M.S.(Gen.Sur)

Department : Department of General Surgery
Government Stanley Medical College,
Chennai-10

The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 13.06.2013 at the Council Hall, Stanley Medical College, Chennai-1 at 2PM

The members of the Committee, the secretary and the Chairman are pleased to approve the proposed work mentioned above, submitted by the principal investigator.

The Principal investigator and their team are directed to adhere to the guidelines given below:

1. You should inform the IEC in case of changes in study procedure, site investigator investigation or guide or any other changes.
2. You should not deviate from the area of the work for which you applied for ethical clearance.
3. You should inform the IEC immediately, in case of any adverse events or serious adverse reaction.
4. You should abide to the rules and regulation of the institution(s).
5. You should complete the work within the specified period and if any extension of time is required, you should apply for permission again and do the work.
6. You should submit the summary of the work to the ethical committee on completion of the work.

19/12/13

MEMBER SECRETARY,
IEC, SMC, CHENNAI

ANNEXURE 2: PROFORMA

"**Comparative Evaluation Of Radiologic And Clinical Scoring Systems In Early Prediction Of Severity Of Acute Pancreatitis"**

Investigator: **Dr.S. VIJAY RAJ**, PG – MS (Gen Surg)

- Name : sl. No:
- Age /sex:
- Address with contact number:
- Ip no:
- Date of admission:
- Date of surgery:

HISTORY OF PRESENTING ILLNESS:

Pain : duration , location , character

Vomiting: onset : duration:

Nausea

Fever

Jaundice

Any other relevant h/o

PAST HISTORY:

WHETHER A KNOWN CASE OF DM/HYPERTENSION/ASTHMA/TB/EPILEPSY/CARDIAC ILLNESS

H/O SIMILAR EPISODES IN THE PAST, IF ANY:

CLINICAL EXAMINATION:

GENERAL EXAMINATION: TEMP: P.R: B.P: R.R

SYSTEMIC EXAMINATION:

CVS

RS

PER ABDOMEN:

CLINICAL DIAGNOSIS:

Investigations:

HEMAT			**LFT**		
HB			T.BIL		
PCV			D.BIL		
RBC			AST		
TC			ALT		
DC			ALP		
PLT			T.PROTEIN		
ESR			S.ALB		
RBS S.AMYLASE S. LIPASE			P.T - T/C INR		
B.UREA			LDH		
S.CREAT			S.CALCIUM		
S.Na+			ABG		
S.K+			BUN		
S.Cl-			**BL.GROUP**		
S.HCO3-					

CHEST X RAY :

ABD X RAY:

USG ABD:

PLAIN CT / CECT ABD

PATIENT CLINICAL COURSE:

OUTCOME OF TREATMENT:

MODIFIED GLASGOW SCORE:

Pa02	<60mm Hg	1
	>60mm Hg	0
AGE	>55 yrs	1
	<55 yrs	0
NEUTROPHILLIA	>15000	1
S. CALCIUM	<8 mg/dl	1
	>=8 mg/dl	0
S. UREA NITROGEN	>45 mg/dl	1
	<= 45 mg/dl	0
S. LDH	> 600 U/l	1
	< = 600 U/l	0
S. ALBUMIN	<3.2 g/dl	1
	>3.2g/dl	0
BLOOD SUGAR	>180 mg/dl	1
	<= 180 mg/dl	0

BISAP

- Blood urea nitrogen (BUN) >25 mg / dl.
- Impaired mental status (GCS < 15).
- SIRS.
- Age >60 years.
- Pleural effusion.

SIRS is defined by presence of two or more of the following criteria:

- Pulse rate > 90/min.
- Respiratory rate > 20/min or PaCO 2 < 32 mm Hg.
- Temperature >100.4 F or < 96.8 F / < 36 or > 38 ° C.
- WBC count >12,000 or < 4,000 cells/$mm^{3,}$ or presence of more than 10% immature blasts.

Modified Glasgow score > 3 indicates severity

BISAP > 3 indicates severity

CT SEVERITY INDEX WITH BALTHAZAR GRADE:

Prognostic Indicator	Points	Grade
Pancreatic inflammation		
Normal pancreas	0	A
Focal or diffuse enlargement of the pancreas	1	B
Intrinsic pancreatic abnormalities with inflammatory changes in peripancreatic fat	2	C
Single, ill-defined fluid collection or phlegmon	3	D
Two or more poorly defined collections or presence of gas in or adjacent to the pancreas	4	E
Pancreatic necrosis		
None	0	
≤ 30%	2	
> 30–50%	4	
> 50%	6	

A COMPARATIVE EVALUATION OF RADIOLOGIC AND CLINICAL SCORING SYSTEMS IN EARLY PREDICTION OF SEVERITY IN ACUTE PANCREATITIS

Investigator: **Dr.Vijay Raj**, PG – MS (Gen Surg).

Patient Information Module

You are being invited to be a subject in this study.

Before you participate in this study, I am giving you the following details about this trial, which includes the aims, methodology, intervention, possible side effects, if any and outcomes:

All patients diagnosed with pancreatitis on clinical grounds or imaging will be included in this study. A detailed clinical history will be taken following a standardized proforma. A detailed clinical examination will be made and relevant investigations, basic and special investigations will be done at the time of admission. CT scan will be done at the time of admission. Efficacy between clinical and radiological scoring systems in predicting severity of pancreatitis will be analysed. The results arising from this study will be analyzed and used for academic purposes. You will be given clear instructions at every step and you are free to ask/ clarify any doubts. Your identity will remain confidential. You are free to withdraw from this trial at any point of time, without any prior notice &/ or without any medical or legal implications.

I request you to volunteer for this study.

Thanking You,

Investigator's Sign Patient's Sign

(Dr.S.VIJAY RAJ) Name:

தீவிர கணைய அழற்சியினால் ஏற்படும் கடுமையினை விரைவில் வருவதுரைத்தல் நுண்கதிரியில் பரிசோதனையா அல்லது மருந்தியக்கச் சோதனையா என ஒப்பிட்டு மதிப்பிடும் ஆய்வு.

ஆய்வாளர் : டாக்டர் ச.விஜய்ராஜ்
முதுநிலை பட்டமேற்படிப்பு மாணவர்
அறுவை சிகிச்சை பட்டப்படிப்பு

வழிகாட்டி : பேராசிரியர் டாக்டர் டார்வின்
அறுவை சிகிச்சை பேராசிரியர்
அரசு ஸ்டான்லி மருத்துவமனை

பங்கேற்பாளரின் தகவல் படிவம்

நீங்கள் இந்த ஆய்வில் பங்கேற்க அழைக்கப்படுகிறீர்கள்.

இந்த ஆய்வில் பங்கேற்கும் முன்னர் இதன் நோக்கத்தையும் முறைகளையும் இதனால் ஏற்படக்கூடிய பின்விளைவுகள் ஏதேனையும் நீங்கள் அறிந்துக் கொள்ள ஆய்வாளர் அளிக்கும் தகவல் பின்வருமாறு

தீவிர கணைய அழற்சியினால் பாதிக்கப்பட்ட நோயாளிகள் மட்டுமே இந்த ஆய்வில் எடுத்துக் கொள்ளப்படுவீர்கள். உங்கள் நோயின் முழு வரலாறும். உங்களின் முழு உடல் பரிசோதனையும் தெளிவாகவும் விரிவாகவும் பதிவு செய்யப்படும். அடிப்படை இரத்த பரிசோதனை மற்றும் நுண்கதிரியியல் பரிசோதனைகளின் முடிவுகள் ஏற்றவாறு பதியப்படும். பரிசோதனைக்கு முன்னும் பின்னும் மற்றும் பரிசோதனையின் பொழுதும் உங்களிடம் ஏற்படும் உடல்நிலை மாற்றங்கள் பதிவு செய்யப்படும் .

இந்த ஆய்வின் முடிவுகள் மருத்துவ காரணங்களுக்காகவும் மருத்துவ கல்விக்காகவும் பயன்படுத்தப்படும். இந்த ஆய்வு பற்றிய சந்தேகங்களுக்கு உரிய முறையில் விளக்கம் அளிக்கப்படும். தங்களை பற்றிய தகவல்கள் இரகசியமாக பாதுகாக்கப்படும்.

இந்த ஆய்வில் இருந்து எப்போது வேண்டுமானாலும் தாங்கள் எவ்வித முன்னறிவிப்பின்றியும், எவ்வித சட்ட சிக்கலும் இன்றியும் விலகிக்கொள்ளலாம். இந்த ஆய்வில் பங்கேற்குமாறு கேட்டுக் கொள்கிறேன்.

நன்றி

ஆய்வாளர் கையொப்பம் நோயாளியின் கையொப்பம்

டாக்டர்.ச.விஜய்ராஜ் (பெயர்)

A COMPARATIVE EVALUATION OF RADIOLOGIC AND CLINICAL SCORING SYSTEMS IN EARLY PREDICTION OF SEVERITY IN ACUTE PANCREATITIS

Investigator: **Dr. S.VIJAY RAJ**, PG – MS (Gen Surg)

Informed Consent

Name: Age/ Sex:

IP:

I herewith declare that I have been explained in a language fully understood by me regarding the purpose of this study, methodology, proposed intervention, plausible side effects, if any and sequelae.

I have been given an opportunity to discuss my doubts and I have received the appropriate explanation.

I understand that my participation in this study is completely voluntary and that I am free to withdraw from this study at anytime without any prior notice &/ or without having my medical or legal rights affected.

I permit the author and the research team full access to all my records at any point, even if I have withdrawn from the study. However my identity will not be revealed to any third party or publication.

I herewith permit the author and the research team to use the results and conclusions arising from this study for any academic purpose, including but not limited to dissertation/ thesis or publication or presentation in any level.

Therefore, in my full conscience, I give consent to be included in the study and to undergo any investigation or any intervention therein.

Patient's Sign Investigator's Sign

(Dr.VIJAY RAJ)

தீவிர கணைய அழற்சியினால் ஏற்படும் கடுமைமினை விரைவில் வருவதுரைத்தல் நுண்கதிரியில் பரிசோதனையா அல்லது மருந்தியக்கச் சோதனையா என ஒப்பிட்டு மதிப்பிடும் ஆய்வு.

ஆய்வாளர் : டாக்டர் ச.விஜய்ராஜ்
முதுநிலை பட்டமேற்படிப்பு மாணவர்
அறுவை சிகிச்சை பட்டப்படிப்பு

வழிகாட்டி : பேராசிரியர் டாக்டர் டார்வின்
அறுவை சிகிச்சை பேராசிரியர்
அரசு ஸ்டான்லி மருத்துவமனை

சுய ஒப்புதல் படிவம்

பெயர் வயது உள்ளிருப்பு எண்:

இந்த மருத்துவ ஆய்வின் விவரங்கள் எனக்கு விளக்கப்பட்டது என்னுடைய சந்தேகங்களை கேட்கவும் அதற்கான தகுந்த விளக்கங்களை பெறவும் வாய்பளிக்கப்பட்டது.

நான் இவ்வாய்வில் தன்னிச்சையாகத் தான் பங்கேற்கிறேன். எந்த காரணத்தினாலும், எந்த கட்டத்திலும் எந்த சட்ட சிக்கலுக்கும் உட்படாமல் இந்த ஆய்விலிருந்து விலகிக் கொள்ளலாம் என்றும் அறிந்துக் கொண்டேன்.

நான் ஆய்விலிருந்து விலகிக் கொண்டாலும் ஆய்வாளர் என்னுடைய மருத்துவ அறிக்கைகளை பார்ப்பதற்கோ அல்லது உபயோகிக்கவோ என் அனுமதி தேவையில்லை என அறிந்துக் கொள்கிறேன். என்னை பற்றிய தகவல்கள் இரகசியமாக பாதுகாக்கப்படும் என்பதை அறிவேன்.

இந்த ஆய்வின் மூலம் கிடைக்கும் தகவல்களையும் பரிசோதனை முடிவுகளையும் ஆய்வாளர் அவர் விருப்பத்திற்கேற்ப எவ்விதமாக பயன்படுத்திக் கொள்ளவும் அதனை பிரசுரிக்கவும் என் முழுமனதுடன் சம்மதிக்கிறேன்.

இந்த ஆய்வில் பங்குக் கொள்ள ஒப்புக் கொள்கிறேன். எனக்கு கொடுக்கப்பட்ட அறிவுரையின்படி நடந்துக் கொள்வதுடன் ஆய்வாளருக்கு உண்மையுடன் இருப்பேன் என்றும் உறுதியளிக்கின்றேன். என் உடல் நலம் பாதிக்கப்பட்டாலோ அல்லது எதிர்பாராத வழக்கத்திற்கு மாறான நோய்குறி தென்பட்டாலோ உடனே அதை தெரிவிப்பேன் என உறுதி கூறுகிறேன்.

இந்த ஆய்வில் எனக்கு எவ்வித மற்றும் அனைத்து பரிசோதனைகளையும் சிகிச்சைகளையும் மேற்கொள்ள நான் முழுமனதுடன் சம்மதிக்கிறேன்.

இப்படிக்கு

ஆய்வாளர் கையொப்பம் நோயாளியின் கையொப்பம்

டாக்டர்.ச.விஜய்ராஜ் (பெயர்)

MASTER CHART

S.NO	NAME	I.P NO	AGE	SEX	PAIN	VOMITING	FEVER	JAUNDICE	ETIOLOGY	GLASGOW	BISAP	CTSI	OUTCOME	COMPLICATIO	MANAGEMENT
1	RAJENDRAN	35399	44	MALE	*	*	NIL	NIL	ALCOHOL	1	2	1	ALIVE		CONSERVATIVE
2	ISRATH	34332	40	FEMALE	*	NIL	NIL	NIL	GALL STONE	2	2	1	ALIVE		CONSERVATIVE
3	MUTHU	34612	52	MALE	*	NIL	*	NIL	ALCOHOL	5	4	3	ALIVE	ARF	CONSERVATIVE
4	GNANVEL	30077	39	MALE	*	NIL	NIL	NIL	ALCOHOL	0	2	2	ALIVE		CONSERVATIVE
5	NAGAPPAN	16628	26	MALE	*	NIL	*	*	IDIOPATHIC	1	2	1	ALIVE		CONSERVATIVE
6	MIJAZ MOHD.	26301	43	MALE	*	NIL	NIL	NIL	ALCOHOL	2	4	2	ALIVE	ARDS	CONSERVATIVE
7	SHANTHA	66982	64	FEMALE	*	NIL	NIL	NIL	GALL STONE	8	5	8	DEAD	PANC. NECROSIS WITH MODS	CONSERVATIVE
8	ARUN	18441	52	MALE	*	NIL	*	NIL	IDIOPATHIC	0	3	2	ALIVE		CONSERVATIVE
9	VINOTH	23987	28	MALE	*	NIL	NIL	*	ALCOHOL	2	2	1	ALIVE		CONSERVATIVE
10	CHINNAPAIYAN	6918	48	MALE	*	*	NIL	NIL	ALCOHOL	1	4	6	DEAD	PANC. NECROSIS	CONSERVATIVE
11	JANARDHANAN	21635	26	MALE	*	NIL	*	*	ALCOHOL	1	3	2	ALIVE		CONSERVATIVE
12	DAMODARAN	39206	31	MALE	*	NIL	*	NIL	GALL STONE	3	3	3	ALIVE	PSEUDOCYST	SURGERY
13	ALBERT	22530	30	MALE	*	*	NIL	*	ALCOHOL	1	2	1	ALIVE		CONSERVATIVE
14	ABDUL QADER	7989	30	MALE	*	NIL	*	*	ALCOHOL	0	3	1	ALIVE		CONSERVATIVE
15	SUBRAMANI	6923	33	MALE	*	NIL	*	NIL	ALCOHOL	1	2	1	ALIVE		CONSERVATIVE
16	VENKATESAN	17439	20	MALE	*	*	*	*	GALL STONE	6	4	3	ALIVE	ARF	CONSERVATIVE
17	BISHNU	320761	45	MALE	*	*	*	NIL	ALCOHOL	1	3	3	ALIVE	PSEUDOCYST	SURGERY
18	FATHIMA	26665	33	FEMALE	*	*	*	NIL	IDIOPATHIC	2	2	2	ALIVE		CONSERVATIVE
19	SENTAMIL SELVI	37807	48	FEMALE	*	NIL	*	NIL	GALL STONE	3	4	6	AMA	PANC. NECROSIS	CONSERVATIVE
20	THILAI NATHAN	36395	34	MALE	NIL	NIL	*	NIL	ALCOHOL	0	2	1	ALIVE		CONSERVATIVE
21	RAJA	37386	54	MALE	*	NIL	*	NIL	ALCOHOL	1	2	1	ALIVE		CONSERVATIVE
22	VIJAYAKUMAR	39214	36	MALE	*	NIL	*	NIL	ALCOHOL	0	1	1	ALIVE		CONSERVATIVE
23	SENTHILVEL	39206	55	MALE	*	NIL	*	NIL	ALCOHOL	4	4	6	ALIVE	PANC. NECROSIS	SURGERY
24	SURYA	39216	44	MALE	*	*	*	*	ALCOHOL	6	4	8	DEAD	PANC. NECROSIS	CONSERVATIVE
25	NALLAKANNU	56890	29	MALE	*	NIL	NIL	NIL	IDIOPATHIC	0	2	1	ALIVE		CONSERVATIVE
26	THANGARASU	37469	52	MALE	NIL	NIL	NIL	NIL	ALCOHOL	1	2	1	ALIVE		CONSERVATIVE
27	SHANTHI	37419	60	FEMALE	*	NIL	*	*	GALL STONE	3	5	3	ALIVE	MODS - SEPSIS	CONSERVATIVE
28	GURU	49947	40	MALE	*	NIL	NIL	NIL	ALCOHOL	1	3	3	ALIVE	PSEUDOCYST	CONSERVATIVE
29	DAS	20029	28	MALE	NIL	NIL	NIL	NIL	ALCOHOL	1	2	1	ALIVE		CONSERVATIVE
30	GEORGE	38435	59	MALE	*	NIL	*	NIL	ALCOHOL	1	2	1	ALIVE		CONSERVATIVE
31	ANGAMMA	42262	27	FEMALE	*	NIL	NIL	NIL	HYPER TGL	3	3	2	ALIVE	HYPOCALCEMIA	CONSERVATIVE
32	VADIVEL	40677	38	MALE	*	*	NIL	NIL	IDIOPATHIC	0	2	1	ALIVE		CONSERVATIVE
33	PANDIYAN	42845	33	MALE	NIL	NIL	*	*	ALCOHOL	1	2	1	ALIVE		CONSERVATIVE
34	MAHARANI	64784	37	FEMALE	*	*	*	NIL	ALCOHOL	0	2	1	ALIVE		CONSERVATIVE
35	KAMARAJ	42947	31	MALE	*	*	*	NIL	ALCOHOL	0	1	1	ALIVE		CONSERVATIVE
36	PERIYASAMY	34459	42	MALE	*	NIL	*	*	ALCOHOL	2	4	6	ALIVE	PANC. NECROSIS	CONSERVATIVE
37	BHARATHI	34520	46	FEMALE	NIL	*	NIL	NIL	GALL STONE	2	3	2	ALIVE		CONSERVATIVE
38	KUPPAN	34781	48	MALE	*	*	NIL	NIL	ALCOHOL	0	2	1	ALIVE		CONSERVATIVE
39	FAROOQ	33291	41	MALE	*	*	*	NIL	ALCOHOL	2	3	1	ALIVE		CONSERVATIVE
40	RAVI	33413	49	MALE	*	NIL	*	NIL	IDIOPATHIC	1	2	1	AMA		CONSERVATIVE
41	RAJAN	30601	51	MALE	*	NIL	*	NIL	IDIOPATHIC	1	4	3	ALIVE	PSEUDOCYST	SURGERY
42	SINGARAM	33744	29	MALE	*	*	NIL	NIL	ALCOHOL	2	1	1	ALIVE		CONSERVATIVE
43	JEEVA	12075	56	MALE	NIL	NIL	*	NIL	ALCOHOL	2	2	1	ALIVE		CONSERVATIVE
44	HARI	32520	33	MALE	*	NIL	NIL	NIL	DRUG	5	4	3	AMA	ARF	CONSERVATIVE
45	DURAI	28928	49	MALE	*	NIL	*	NIL	ALCOHOL	0	4	1	DEAD	SEPSIS	CONSERVATIVE
46	JOSEPH	30619	27	MALE	*	NIL	*	NIL	IDIOPATHIC	1	0	1	ALIVE		CONSERVATIVE
47	RADHA	88937	39	FEMALE	NIL	*	NIL	NIL	GALL STONE	0	2	1	ALIVE		CONSERVATIVE
48	SAMIKANNU	59188	28	MALE	*	*	*	NIL	ALCOHOL	1	3	2	ALIVE		CONSERVATIVE
49	KUTTIYAMMAL	19136	36	FEMALE	*	*	NIL	NIL	ALCOHOL	2	2	1	ALIVE		CONSERVATIVE
50	BALU	26621	28	MALE	*	NIL	*	NIL	ALCOHOL	0	2	2	ALIVE		CONSERVATIVE